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SEARCH REQUEST FORM

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Requester's Full Name: Lisa V. Cook Examiner #: 77134 Date: 3/30/01
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Title of Invention: Method for examining Kidney Disease

Inventors (please provide full names): masaya Yamamouchi, aKio Honda, Hiromi Hase, Takeshi Sugaya, Kenjiro Kimura

Earliest Priority Filing Date: 11/26/1998

For Sequence Searches Only *Please include all pertinent information (parent, child, divisional, or issued patent numbers) along with the appropriate serial number.*

Please also see attached bib sheet + claims.

Key words:
kidney disease - kidney/renal failure
nephrology
renal replacement therap?
blood proteins
macroglobulins insufficien?
1) α2u-globulin (alpha 2 macro globulin) also known as major urinary protein
2) GlmB (GBM in disclosure) - mouse glomilar basal membrane)
3) fatty acid binding protein (FABP) or renal tissue or kidney/renal/proximal tubule or urine or liver type
examin detect? diagnos identif analy? analy?

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Thanks
1446 1435 Lisa Cook

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Type of Search

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Structure (#)
Bibliographic ✓
Litigation
Fulltext
Patent Family
Other

Vendors and cost where applicable

STN 26.3.01
Questel/Orbit
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Lexis/Nexis
Sequence Systems
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Other (specify)

PTO-1590 (1-2000)

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                 Derwent World Patents Index files
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NEWS 11  Dec 17  Merged CEABA-VTB for chemical engineering and
                 biotechnology
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L2 ANSWER 1 OF 23 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1999:9518 BIOSIS
DN PREV199900009518
TI Decorin deficiency accelerates extracellular matrix (ECM) accumulation in anti-glomerular basement membrane (**anti-GMB**) **nephritis**.
AU Ha, Il Soo (1); Iozzo, Renato V.; Noble, Nancy A.; Border, Wayne A.
CS (1) Univ. Utah Sch. Med., Salt Lake City, UT USA
SO Journal of the American Society of Nephrology, (Sept., 1998) Vol. 9, No. PROGRAM AND ABSTR. ISSUE, pp. 516A.
Meeting Info.: 31st Annual Meeting of the American Society of Nephrology Philadelphia, Pennsylvania, USA October 25-28, 1998 American Society of Nephrology
. ISSN: 1046-6673.
DT Conference
LA English
CC Immunology and Immunochemistry - General; Methods *34502
Cytology and Cytochemistry - Animal *02506
Metabolism - Metabolic Disorders *13020
Urinary System and External Secretions - General; Methods *15501
General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals *00520
Biochemical Studies - General *10060
BC Muridae 86375
IT Major Concepts
Immune System (Chemical Coordination and Homeostasis); Urinary System (Chemical Coordination and Homeostasis)
IT Parts, Structures, & Systems of Organisms
extracellular matrix
IT Diseases
anti-glomerular basement membrane **nephritis**: immune system disease, urologic disease; decorin deficiency: metabolic disease
IT Alternate Indexing
Anti-Glomerular Basement Membrane Disease (MeSH)
IT Miscellaneous Descriptors
Meeting Abstract; Meeting Poster
ORGN Super Taxa
Muridae: Rodentia, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
mouse (Muridae): strain-DKO
ORGN Organism Superterms
Animals; Chordates; Mammals; Nonhuman Mammals; Nonhuman Vertebrates; Rodents; Vertebrates

L2 ANSWER 2 OF 23 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1989:136558 BIOSIS
DN BA87:71211
TI IDENTIFICATION OF GOODPASTURE ANTIGENS IN HUMAN ALVEOLAR BASEMENT MEMBRANE.
AU YOSHIOKA K; ISEKI T; OKADA M; MORIMOTO Y; ERYU N; MAKI S
CS DEP. PEDIATRICS, KINKI UNIV. SCH. MED., 377-2, OHNO-HIGASHI, OSAKA-SAYAMA 589, JAPAN.
SO CLIN EXP IMMUNOL, (1988) 74 (3), 419-424.
CODEN: CEXIAL. ISSN: 0009-9104.
FS BA; OLD
LA English
AB Goodpasture (GP) antigens, protein components reactive with human autoantibodies against glomerular basement membrane (GBM), were identified in human alveolar basement membrane (ABM) using an enzyme-linked immunoassay (ELISA), Western blotting and immunoprecipitation. All six anti-GBM antisera studied, three obtained form patients with glomerulonephritis and pulmonary haemorrhages (I.e. GP syndrome), and three from patients with glomerulonephritis alone, distinctively reacted with collagenase-digested (CD) ABM. Very cationic 22-28 kD and 40-48 kD components were detected by blot analysis combined with two-dimensional gel electrophoresis. These proteins showed some similarities to GP antigens in human GMB with respect to the monomer-dimer composition and charge distribution. Inhibition ELISA revealed that the binding of **anti-GMB** antisera to CDGBM decreased when they were pre-incubated with CDABM, suggesting that the anti-GBM antisera recognized the same epitope(s) on the GBM and ABM. Heterogeneity of the GP antigens in human ABM was demonstrated by blotting: monomeric antigens were absent

Disease *12508
Cardiovascular System - Blood Vessel Pathology *14508
Urinary System and External Secretions - Pathology *15506
Respiratory System - General; Methods 16001
Respiratory System - Pathology *16006
Immunology and Immunochemistry - General; Methods *34502
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
BC Hominidae 86215
IT Miscellaneous Descriptors
GLOMERULONEPHRITIS PULMONARY HEMORRHAGE LUNG INVOLVEMENT AUTOANTIBODY PROTEIN COMPONENT MONOMER-DIMER COMPOSITION VARIATION ELISA WESTERN ROOT IMMUNOPRECIPITATION

L2 ANSWER 3 OF 23 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1985:302346 BIOSIS
DN BA79:82342
TI DETECTION OF TERMINAL COMPLEMENT COMPONENTS IN EXPERIMENTAL IMMUNE GLOMERULAR INJURY.
AU ADLER S; BAKER P J; PRITZL P; COUSER W G
CS DIV. NEPHROL., BOX RM-11, UNIV. WASHINGTON, SEATTLE, WASH. 98195, U.S.A.
SO KIDNEY INT, (1984 (RECD 1985)) 26 (6), 830-837.
CODEN: KDYIA5. ISSN: 0085-2538.
FS BA; OLD
LA English
AB Complement mediates glomerulonephritis by inflammatory cell-dependent and non-inflammatory cell-independent effects on glomerular permeability. The latter may involve terminal components of the complement system. Several models of immunologic renal injury were examined in the rat by immunofluorescence (IF) for terminal complement components C5, C6, C7 and C8 in glomeruli using antisera to human C5-8, which cross-react with the analogous rat complement components. Rats with the heterologous and autologous phases of passive Heymann **nephritis** (PHN) had proteinuria and 1 to 2+ capillary wall deposits of heterologous or rat IgG, rat C3, and C5-8. Complement depletion with cobra venom factor (CVF) significantly decreased proteinuria in both models and prevented deposition of all complement components. Rats with active Heymann **nephritis** had similar deposits of rat IgG and C5-8. Rats with **anti-GMB** [glomerular basement membrane] **nephritis** and aminonucleoside nephrosis had severe proteinuria which was not affected by CVF treatment and deposits of C5-8 were absent. The presence of terminal complement components in immune deposits in experimental glomerular disease correlates with a functional role for complement in mediating glomerular injury. The terminal complement pathway may be a major mediator of some types of immune glomerular injury.
CC Cytology and Cytochemistry - Animal *02506
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Biochemical Studies - Carbohydrates 10068
Biophysics - General Biophysical Techniques 10504
Pathology, General and Miscellaneous - Inflammation and Inflammatory Disease *12508
Metabolism - Carbohydrates *13004
Metabolism - Proteins, Peptides and Amino Acids *13012
Metabolism - Metabolic Disorders *13020
Urinary System and External Secretions - General; Methods 15501
Urinary System and External Secretions - Pathology *15506
Immunology and Immunochemistry - General; Methods 34502
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
BC Muridae 86375
IT Miscellaneous Descriptors
RAT PASSIVE HEYMANN **NEPHRITIS** IMMUNOGLOBULIN PROTEINURIA IMMUNOFLUORESCENCE

L2 ANSWER 4 OF 23 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1984:347495 BIOSIS
DN BA78:83975
TI ASSOCIATION OF IMMUNO GLOBULIN GM ALLOTYPES WITH ANTI GLOMERULAR BASEMENT MEMBRANE ANTIBODIES AND THEIR TITER.
AU REES A J; DEMAINE A G; WELSH K I
CS DEP. MED., ROYAL POSTGRAD. MED. SCH., HAMMERSMITH HOSP., DUCANE RD., LONDON W12, UK.
SO HUM IMMUNOL, (1984) 10 (4), 213-220.

influence susceptibility to or clinical expression of **anti-GMB** disease.

CC Genetics and Cytogenetics - Human *03508
Genetics and Cytogenetics - Population Genetics 03509
Clinical Biochemistry; General Methods and Applications *10006
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Biochemical Studies - Carbohydrates 10068
Pathology, General and Miscellaneous - Inflammation and Inflammatory Disease 12508
Metabolism - Carbohydrates *13004
Metabolism - Proteins, Peptides and Amino Acids *13012
Urinary System and External Secretions - Pathology *15506
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508

BC Hominidae 86215

IT Miscellaneous Descriptors
HUMAN CAUCASIAN GENETIC SUSCEPTIBILITY GLOMERULAR **NEPHRITIS**

L2 ANSWER 5 OF 23 BIOSIS COPYRIGHT 2001 BIOSIS

AN 1979:206946 BIOSIS

DN BA68:9450

TI GLOMERULO **NEPHRITIS** AUTO IMMUNITY AUTO ANTIBODY.

AU BANKS K L

CS DEP. VET. MICROBIOL. PATHOL., WASH. STATE UNIV., PULLMAN, WASH. 99164, USA.

SO AM J PATHOL, (1979) 94 (2), 443-446.
CODEN: AJPAA4. ISSN: 0002-9440.

FS BA; OLD

LA English

AB Horses (3) are presented with glomerular basement membrane (GMB) disease with renal failure mimicking human glomerulonephritis. Kidney tissues are examined at autopsy revealing **anti-GMB** antibody by fluorescein light microscopy and EM. Presence of autoimmune disease is verified by glomerular immunoglobulin and complement (C3) associated complexes.

CC Microscopy Techniques - General and Special Techniques 01052
Microscopy Techniques - Electron Microscopy 01058
Biochemical Studies - General 10060
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Biochemical Studies - Carbohydrates 10068
Biophysics - Membrane Phenomena 10508
Pathology, General and Miscellaneous - Comparative 12503
Pathology, General and Miscellaneous - Inflammation and Inflammatory Disease 12508
Pathology, General and Miscellaneous - Necrosis 12510
Cardiovascular System - Blood Vessel Pathology 14508
Blood, Blood-Forming Organs and Body Fluids - Blood and Lymph Studies 15002
Urinary System and External Secretions - General; Methods 15501
Urinary System and External Secretions - Anatomy 15502
Urinary System and External Secretions - Pathology *15506
Immunology and Immunochemistry - General; Methods 34502
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
Veterinary Science - General; Methods 38002
Veterinary Science - Pathology *38004

BC Equidae 86145
Hominidae 86215

IT Miscellaneous Descriptors
HORSE HUMAN RENAL FAILURE IMMUNO GLOBULIN COMPLEMENT ELECTRON MICROSCOPY LIGHT MICROSCOPY AUTOPSY

L2 ANSWER 6 OF 23 CAPLUS COPYRIGHT 2001 ACS

AN 1983:593018 CAPLUS

DN 99:193018

TI Glomerular prostaglandin and thromboxane synthesis in rat nephrotoxic serum **nephritis**. Effects on renal hemodynamics

AU Lianos, Elias A.; Andres, Giuseppe A.; Dunn, Michael J.

CS Dep. Med., Case West. Reserve Univ., Cleveland, OH, 44106, USA

SO J. Clin. Invest. (1983), 72(4), 1439-48
CODEN: JCINAO; ISSN: 0021-9738

DT Journal

LA English

GFR and RPF coincided with increments in vasodilatory PG, (PGE2 and PGI2). The thromboxane synthetase inhibitor OKY-1581 markedly inhibited platelet and glomerular TXB2 synthesis and preserved GFR at 1, 2, 3 h. Another thromboxane synthetase inhibitor, UK-38485, also completely inhibited platelet and glomerular TXB2 synthesis and prevented decrement of GFR at 2 and 3 h. A cycloocygenase inhibitor, ibuprofen, inhibited platelet TXB2 and PGE2 synthesis and reduced glomerular PGE2 but not TXB2 synthesis. In the ibuprofen-treated rats, the partial recoveries of GFR and RPF at 3 h were attenuated. The in vitro glomerular TXB2 synthesis correlated inversely with the presacrifice GFR and filtration fraction. Apparently, in anti-GBM **nephritis** there is enhanced synthesis of TXA2 and PG in the glomerulus that mediate changes in renal hemodynamics.

ST prostaglandin thromboxane nephrotoxic serum **nephritis**; hemodynamics kidney nephrotoxic **nephritis** prostanoid

IT Prostaglandins
RL: FORM (Formation, nonpreparative)
(formation of, by glomerulus in nephrotoxic serum **nephritis**)

IT Circulation
(of kidney, prostaglandin and thromboxane formation by glomerulus in nephrotoxic serum **nephritis** in relation to)

IT Kidney, disease or disorder
(immune complex glomerulonephritis, prostaglandin and thromboxane formation by glomerulus in)

IT 363-24-6 551-11-1 35121-78-9 57576-52-0 58962-34-8
RL: FORM (Formation, nonpreparative)
(formation of, by glomerulus in nephrotoxic serum **nephritis**)

L2 ANSWER 7 OF 23 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.

AN 95000051 EMBASE

DN 1995000051

TI Contribution of ED-1- and CD-8-positive cells to the development of crescentic-type anti-GBM **nephritis** in rats.

AU Hattori T.; Nagamatsu T.; Ito M.; Suzuki Y.

CS Department of Pharmacology, Faculty of Pharmacy, Meijo University, 150 Yagotoyama,Nagoya 468, Japan

SO Japanese Journal of Nephrology, (1994) 36/11 (1228-1239).
ISSN: 0385-2385 CODEN: NJGKAU

CY Japan

DT Journal; Article

FS 026 Immunology, Serology and Transplantation
028 Urology and Nephrology
037 Drug Literature Index

LA English

SL English

AB The current studies were designed to identify which mononuclear leukocytes have an important role in the development of glomerular injury using rats with orginal-type (mild injury) and crescentic-type (severe injury) anti-glomerular basement membrane (GBM) **nephritis**. 1) Proteinuria was persistent in crescentic-type anti-GBM **nephritis** compared with orginal-type anti-GBM **nephritis**. Macrophages/moncytes (ED-1), cytotoxic/suppressor T cells (CD-8), interleukin-2-receptor (CD-25)-positive cells and Ia-positive cells accumulated remarkably and persisted for longer in crescentic-type nephritic glomeruli. 2) We then performed investigations using immunosuppressants. Cyclosporin A abrogated proteinuria more effectively than azathioprine in crescentic-type **nephritis**. However, plasma antibody titer and glomerular rat IgG deposition were equally reduced by both azathioprine and cyclosporin A. The increase in the numbers of ED-1-, CD-8- and CD-25-positive cells in nephritic glomeruli was completely inhibited by cyclosporin A, but inhibited only slightly by azathioprine. 3) There was a correlation between the degree of proteinuria and the number of ED-1- and CD-8-positive cells. It is likely that these cells are leukocytes that lead to glomerular injury in **nephritis**. 4) In additional experiments using monoclonal antibodies against macrophages/monocytes and cytotoxic/suppressor T cells, urinary protein excretion and accumulation of these cells were blunted in nephritic rats treated with these antibodies. These results suggest that ED-1- and CD-8-positive cells are involved in the development of crescentic-type **anti-GMB nephritis**.

CT Medical Descriptors:
*glomerulonephritis: ET, etiology
*kidney injury: ET, etiology

CO Sandoz (Switzerland); Sigma (United States)

L2 ANSWER 8 OF 23 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 89245706 EMBASE
DN 1989245706
TI The development of anti-glomerular basement membrane **nephritis** in two children with Alport's syndrome after renal transplantation: Characterization of the antibody target.
AU d. Heuvel V.L.P.W.J.; Schroder C.H.; Savage C.O.S.; Menzel D.; Assmann K.J.M.; Monnens L.A.H.; Veerkamp J.H.
CS Department of Biochemistry, University of Nijmegen, 6500 HB Nijmegen, Netherlands
SO Pediatric Nephrology, (1989) 3/4 (406-413).
ISSN: 0931-041X CODEN: PEDNEF
CY Germany
DT Journal
FS 005 General Pathology and Pathological Anatomy
007 Pediatrics and Pediatric Surgery
028 Urology and Nephrology
LA English
SL English
AB Two children with Alport's syndrome are described, who developed anti-glomerular basement membrane (GBM) antibody-mediated **nephritis** after renal transplantation. The reactivity of antibodies in their serum with collagenase-solubilized normal GBM was examined by SDS-PAGE with one- and two-dimensional immunoblotting. The specificity was compared with that of antibodies present in serum from a patient with Goodpasture's syndrome, and a mouse monoclonal antibody (MCA-P1), directed against the Goodpasture antigen. All reacted in a similar way with collagenase-solubilized GBM. Since abnormalities in the composition of the GBM are present in Alport's syndrome, it is proposed that differing antigen composition of GBM in the host compared with the donor kidney, together with transplant rejection, may have provoked the development of post-transplant **anti-GMB** antibodies.
CT Medical Descriptors:
*alport syndrome
*glomerulonephritis
*goodpasture syndrome
*kidney transplantation
adolescent
child
histochemistry
histology
case report
human
male
female
priority journal
complication
Drug Descriptors:
*glomerulus basement membrane antibody

L2 ANSWER 9 OF 23 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 89204518 EMBASE
DN 1989204518
TI Transfer of anti-glomerular basement membrane antibody-induced glomerulonephritis in inbred rats with isologous antibodies from the urine of nephritic rats.
AU Sado Y.; Naito I.; Okigaki T.
CS Division of Immunology, Shigei Medical Research Institute, Yamada, Okayama 701-02, Japan
SO Journal of Pathology, (1989) 158/4 (325-332).
ISSN: 0022-3417 CODEN: JPTLAS
CY United Kingdom
DT Journal
FS 005 General Pathology and Pathological Anatomy
026 Immunology, Serology and Transplantation
028 Urology and Nephrology
LA English
SL English
AB Anti-glomerular basement membrane antibody-induced glomerulonephritis (**anti-GMB nephritis**) was transferred from nephritic rats to normal recipient rats with isologous antibodies obtained

CT Medical Descriptors:
*basement membrane
*glomerulonephritis
*glomerulus
histochemistry
histology
rat
urine
animal experiment
animal cell
nonhuman
priority journal
Drug Descriptors:
antibody

L2 ANSWER 10 OF 23 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 88005699 EMBASE
DN 1988005699
TI Characterisation and specificity of glomerular basement membrane antigens identified by sera of patients with **anti-GMB nephritis**.
AU Wingen A.-M.; Rauterberg E.W.
CS Institute of Immunology and Serology, University of Heidelberg, D-6900 Heidelberg, Germany
SO Nephrology Dialysis Transplantation, (1986) 1/3 (155-163).
ISSN: 0931-0509 CODEN: NDTREA
CY Germany
DT Journal
FS 028 Urology and Nephrology
005 General Pathology and Pathological Anatomy
026 Immunology, Serology and Transplantation
LA English
SL English
AB The sera of 21 patients positive for antibodies against GBM in indirect immunofluorescence tests were examined by immunoblotting. We demonstrated antibodie4s against 50, 48, 43 and 29 kD molecular weight peptides in 20 of 21 sera using collagenase-digested GBM, in 19 of 21 using trypsin-digested GBM, and in 10 of 21 using elastase-digested GBM. Although the spectrum of molecular weights of the antigenic proteins was similar in all three digests, they differed with respect to preservation of antigenicity upon reduction with mercaptoethanol. Many of the sera of patients and controls reacted with proteins unrelated to GBM, e.g. albumin and prealbumin. Furthermore, some control sera reacted with one single peptide of the above-mentioned specific GBM peptides. Our results suggest that the highly purified 29 kD peptide of the collagenase digest or the 50 kD peptide of the trypsin digest provide the best antigens to develop a screening test for antibbodies against GBM. However, serum antibodies against these antigens will not be absolutely specific for **anti-GMB** antibody-mediated **nephritis**, as shown by the immunoblot experiments.
CT Medical Descriptors:
*glomerulonephritis
*glomerulus basement membrane
immunoblotting
human
clinical article
Drug Descriptors:
*glomerulus basement membrane antibody

L2 ANSWER 11 OF 23 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 85024587 EMBASE
DN 1985024587
TI The influence of HLA-linked genes on the severity of **anti-GMB** antibody-mediated **nephritis**.
AU Rees A.J.; Peters D.K.; Amos N.; et al.
CS Medical Research Council Clinical Immunology Research Group, Department of Medicine and Immunology, Royal Postgraduate Medical School, Hammersmith Hospital, London W12 0HS, United Kingdom
SO Kidney International, (1984) 26/4 (444-450).
CODEN: KDYIA5
CY United States
DT Journal
FS 028 Urology and Nephrology

anti-GBM disease. Such an association was probable for patients in group 1 (P = 0.27 x 10-6), likely for those in group 2 (P = 0.024) but unlikely for patients in group 3 (P = 0.62) suggesting HLA-B7-associated genes influence severity. Clinical results from a subset of the patients referred directly on presentation showed that patients who inherited HLA-B7 together with DR2 had significantly higher plasma creatinines, a greater proportion of glomeruli surrounded by crescents and a worse prognosis. Despite this there was little difference in severity of their lung disease.

CT Medical Descriptors:
*glomerulonephritis
kidney
priority journal
heredity
major clinical study
diagnosis
human
Drug Descriptors:
*HLA B7 antigen
*HLA DR2 antigen
*glomerulus basement membrane antibody

L2 ANSWER 12 OF 23 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 85024581 EMBASE
DN 1985024581
TI Effect of antibody charge and concentration on deposition of antibody to glomerular basement membrane.
AU Madaio M.P.; Salant D.J.; Adler S.; et al.
CS Evans Memorial Department of Clinical Research, University Hospital, Boston University Medical Center, Boston, MA, United States
SO Kidney International, (1984) 26/4 (397-403).
CODEN: KDYIA5
CY United States
DT Journal
FS 028 Urology and Nephrology
026 Immunology, Serology and Transplantation
023 Nuclear Medicine
005 General Pathology and Pathological Anatomy
LA English
SL French
AB Fixed anionic sites within the glomerular capillary wall influence the permeation of serum proteins, the localization of various antigens, and the deposition of antibody in the subepithelial space. In anti-GBM **nephritis** antibody deposition occurs very rapidly to antigenic sites located relatively proximal in the glomerular capillary wall. We examined the influence of the glomerular charge barrier on anti-GBM antibody deposition by comparing the rate of deposition of antibodies with cationic and anionic isoelectric points. Purified sheep anti-rat GBM IgG was isolated from acid eluates of kidneys obtained 24 hr after rats were injected with sheep antiserum to rat GBM. Anti-GBM IgG was separated into cationic (pI 6.4-8.5) and anionic (pI 4.2-6.8) fractions, which were radiolabelled with 131I and 125I, respectively, shown to have equal antibody contents measured by in vitro binding to normal glomeruli, mixed in equal amounts, and injected in incremental doses to ten rats. At 1 hr the glomerular antibody binding of each fraction was directly related to the blood level (r = 0.95, r = 0.97) and delivery of antibody (r = 0.98, r = 0.98). Glomerular binding of cationic antibody was four times greater than anionic antibody over the entire range of deliveries studied (P < 0.001). We conclude that glomerular deposition of **anti-GMB** antibody is directly related to blood concentration and delivery of antibody. Furthermore, the deposition of cationic antibodies to GBM antigens was significantly greater than the deposition of anionic antibodies. The charge-selective glomerular filtration barrier may be an important determinant of the quantity and subclass composition of anti-GBM IgG deposits in glomeruli, and therefore of the severity of tissue injury produced.

CT Medical Descriptors:
*glomerulonephritis
*glomerulus basement membrane
*immune complex deposition
***nephritis**
electricity
rat

SO Journal of Clinical Investigation, (1983) 72/4 (1439-1448).
CODEN: JCINAO
CY United States
DT Journal
FS 028 Urology and Nephrology
025 Hematology
023 Nuclear Medicine
LA English
AB Glomerular arachidonate cyclooxygenation by isolated rat glomeruli was assessed in vitro in antiglomerular basement membrane (anti-GBM) antibody-induced glomerulonephritis by radioimmunoassay for prostaglandins (PG) and thromboxane. After a single intravenous injection of rabbit anti-rat GBM serum, we observed enhancement of glomerular thromboxane B2 (TxB2) synthesis as early as 2 to 3 h with smaller increments in PGF(2.alpha.), PGE2 and 6-keto-PGF(2.alpha.) and PGE2 remained enhanced, whereas on days 8, 11, and 14, TxB2 was the only prostanoid synthesized at increased rates. Glomerular TxB2 synthesis correlated with the presacrifice 24-h protein excretion. 60 min after intravenous infusion of **anti-GMB** serum, glomerular filtration rate (GFR) decreased (0.66 .+-. 0.04 to 0.44 .+-. 0.03 ml/min per 100 g, P < 0.05), without a significant change in renal plasma flow (RPF): 1.97 .+-. 0.23 to 1.80 .+-. 0.23 ml/min per 100 g) and without a change in glomerular PG synthetic rates. At 2 h, GFR and RPF reached a nadir (0.25 .+-. 0.04 and 1.3 .+-. 0.1 ml/min per 100 g, respectively) coinciding with a fivefold increment in glomerular TxB2. By 3 h GFR and RPF partially recovered to 0.43 .+-. 0.07 and 1.77 .+-. 0.20 ml/min per 100 g, respectively, P < 0.05, despite further increments in TxB2 synthesis. This recovery of GFR and RPF coincided with increments in vasodilatory PG, (PGE2 and PGI2). The thromboxane synthetase inhibitor OKY-1581 markedly inhibited platelet and glomerular TxB2 synthesis and preserved GFR at 1, 2, and 3 h. Another thromboxane synthetase inhibitor, UK-38485, also completely inhibited platelet and glomerular TxB2 synthesis and prevented decrements of GFR at 2 and 3 h. A cyclooxygenase inhibitor, ibuprofen, inhibited platelet TxB2 and PGE2 synthesis and significantly reduced glomerular PGE2 but not TxB2 synthesis. In the ibuprofen-treated rats, the partial recoveries of GFR and RPF at 3 h were attenuated. The in vitro glomerular TxB2 synthesis correlated inversely with the presacrifice GFR and filtration fraction. These observations indicate that in anti-GBM **nephritis** there is enhanced synthesis of TxA2 and PG in the glomerulus that mediate changes in renal hemodynamics.
CT Medical Descriptors:
*glomerulus
*kidney blood flow
***nephrotoxic serum nephritis**
glomerulonephritis
hemodynamics
kidney
radioimmunoassay
rat
animal experiment
human
Drug Descriptors:
*prostaglandin
*thromboxane
radioisotope
RN (thromboxane) 66719-58-2

L2 ANSWER 14 OF 23 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 80009972 EMBASE
DN 1980009972
TI Crescentic glomerulonephritis without immune deposits: Clinicopathologic features.
AU Stilmant M.M.; Bolton W.K.; Sturgill B.C.; et al.
CS Dept. Pathol., Mallory Inst. Pathol., Boston City Hosp., Boston, Mass., United States
SO Kidney International, (1979) 15/2 (184-195).
CODEN: KDYIA5
CY United States
DT Journal
FS 028 Urology and Nephrology
026 Immunology, Serology and Transplantation
005 General Pathology and Pathological Anatomy
LA English

reported in **anti-GMB** and immune-complex-induced glomerulonephritis. These observations expand the spectrum of rapidly progressive crescentic glomerulonephritis. They suggest that glomerular immune deposits may be less important than other factors in determining the extent of renal injury and subsequent clinical course in crescentic glomerulonephritis.

CT Medical Descriptors:
*rapidly progressive glomerulonephritis
*glomerulus epithelium
*immune complex disease
*proliferative glomerulonephritis
glomerulonephritis
kidney biopsy
major clinical study
histology
cytology
kidney
diagnosis

L2 ANSWER 15 OF 23 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 79003346 EMBASE
DN 1979003346
TI Plasma protein handling in the rat kidney: Micropuncture experiments in the acute heterologous phase of anti-GBM-**nephritis**.
AU Galaske R.G.; Baldamus C.A.; Stolte H.
CS Dept. Innere Med., Med. Hochsch. Hannover, D-3000 Hannover, Germany
SO Pflugers Archiv European Journal of Physiology, (1978) 375/3 (269-277).
CODEN: PFLABK
CY Germany
DT Journal
FS 002 Physiology
028 Urology and Nephrology
LA English
AB Glomerular filtration and tubular uptake of plasma proteins have been studied in the rat using micropuncture techniques. Under control conditions the glomerular capillary wall is an effective barrier, only 7.6 .mu.g/min x 100 g BW albumin have been measured as filtered load. Four to twelve hours after i.v. injection of anti-glomerular-basement membrane serum (**anti-GMB**-serum) sieving coefficient phi and filtered load increased in a dose-dependent manner (phi albumin in controls = 0.27 x 10-3, after injection of 0.5 ml Antiserum phi=0.28 x 10-3 and 1.0 ml Antiserum phi=2.32 x 10-3. The tubular reabsorption capacity is almost reached under control conditions and amounts to 5.6-10.7 .mu.g/min x 100 g BW for albumin. Only reduced GFR (0.36 .+-. 0.07 ml/min x 100 g BW) and reduced tubular flow lead to increased tubular uptake under overload conditions (10.7 vs. 99.0 .mu.g albumin/min x 100 g BW). Tubular reabsorption of so-called high-molecular-weight proteins seems to be a nonselective mechanism. The ratio Alb/Alb + Glob (89.9-93.1%) did not differ significantly at the individual puncture sites and in the final urine.
CT Medical Descriptors:
*glomerulus filtration
*glomerulus filtration rate
*kidney tubule absorption
***nephritis**
*proteinuria
glomerulonephritis
puncture
intravenous drug administration
kidney
animal experiment
rat
Drug Descriptors:
*glomerulus basement membrane antibody
*immunoglobulin
RN (immunoglobulin) 9007-83-4

L2 ANSWER 16 OF 23 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 77127539 EMBASE
DN 1977127539
TI Association of crescentic glomerulonephritis with membranous glomerulonephropathy: a report of three cases.
AU Murphy P.V.; Zimmerman S.W.; Burkholder P.M.; Lagrimas A.J.

glomerulonephropathy. **Anti GMB** antibodies were present in this patient's serum. The third patient presented with acute renal failure of moderate severity. A renal biopsy revealed crescentic **nephritis**, granular deposits of immunoglobulins, and epimembranous electron dense deposits typical of membranous glomerulonephropathy. Although his creatinine clearance improved spontaneously, nephrotic syndrome has persisted and a repeat renal biopsy showed a progression of the membranous glomerulonephropathy with the disappearance of the crescentic lesions. The reason for this peculiar association of membranous glomerulonephropathy and crescentic glomerulonephritis is unclear. It is possible that deposition of immune complexes along glomerular basement membrane may render the glomerulus more susceptible to additional injury from a variety of other agents. Alternatively, deposits formed in one disease could initiate release of normal or altered basement membrane material and lead to formation of anti GBM antibodies and subsequent development of crescentic **nephritis**.

CT Medical Descriptors:
*chronic kidney failure
*glomerulonephritis
*glomerulus
*membranous glomerulonephritis
methodology
histology
major clinical study
diagnosis
electron microscopy
Drug Descriptors:
*glomerulus basement membrane antibody

L2 ANSWER 17 OF 23 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 76080352 EMBASE
DN 1976080352
TI Tuberculin (PPD) reactivity in anti GBM **nephritis**.
AU Couser W.G.; Lewis E.J.
CS Dept. Med., Boston Univ., Boston, Mass., United States
SO Clinical Research, (1975) 23/3 (358A).
CODEN: CLREAS
DT Journal
FS 037 Drug Literature Index
028 Urology and Nephrology
LA English
AB The mechanism of sensitization to glomerular basement membrane (GMB) antigens in patients with **anti GMB** antibody mediated glomerulonephritis is not known. Production of experimental autoimmune anti GBM **nephritis** requires injection of GMB and Freunds adjuvant containing mycobacteria (CFA), and prior sensitization with CFA markedly enhances the nephrotoxicity of heterologous antibody to GMB. The prevalence of hypersensitivity to mycobacterial antigens in **anti GMB nephritis** was evaluated retrospectively in 10 patients with rapidly progressive glomerulonephritis (RPGN) crescents in over 50% of glomeruli and linear deposition of IgG along the GBM. Eight patients had circulating antibody to GMB and 7 had anti GBM antibody deposition confirmed by elution studies. cutaneous hypersensitivity (CH) to 0.02-0.1 .mu.g of PPD was demonstrated in 8/10 (80%) patients by development of > 8 mm of induration at the skin test site in 48 hours. Two patients with typical clinical and pathologic findings were PPD negative. No patient had other clinical evidence of mycobacterial infection. Three patients had a family history of tuberculosis. The prevalence of CH to PPD in these patients differed significantly from that in 42 patients with renal disease of diverse etiologies matched for age, renal function and previous transfusion (7%, $p < 0.01$) and the general population (3-14%, $p < 0.01$). This study demonstrates a significant association between CH to PPD and **anti GMB nephritis** in 1 group of patients. Sensitization to mycobacterial antigens may have an adjuvant effect on the immune response and facilitate development of **anti GMB** antibody mediated RPGN in man.

CT Medical Descriptors:
*clinical study
*glomerulonephritis
*glomerulus
*glomerulus basement membrane
*kidney

CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals; Cancer Journals
EM 199607
AB BACKGROUND: In the absence of evidence of arteritis or Wegener's granulomatosis, the syndrome of lung hemorrhage and **nephritis** has been commonly associated with anti-glomerular basement membrane (GBM) antibodies. However, it has been increasingly recognized that many cases are associated with antineutrophil cytoplasmic antibodies (ANCAs). OBJECTIVE: To review available clinical and pathologic findings to determine the diseases accounting for lung hemorrhage and **nephritis**. METHODS: We studied the records of 750 patients from whom serum samples were sent to our laboratory for anti-GBM antibody assays between 1981 and 1993 and found 88 patients with evidence of lung hemorrhage and **nephritis**. Serum samples were retested, using current methods, for anti-GBM antibodies (against noncollagenous 1 domain of the alpha 3 chain of type IV collagen) and for antibodies to proteinase 3 and myeloperoxidase--the two types of ANCA of diagnostic value. RESULTS: Of 88 patients with evidence of lung hemorrhage and **nephritis**, 48 had ANCAs, six had anti-GBM antibodies, and seven had both. In 48 patients with ANCAs, the pathologic findings that accounted for the pulmonary renal syndrome were pauci-immune necrotizing and crescentic glomerulonephritis and pulmonary capillaritis. Only eight had convincing evidence (during life) of Wegener's granulomatosis and only one other had documented arteritis. In 27 patients without ANCAs or anti-GBM antibodies, a variety of unrelated renal and pulmonary diseases were found. CONCLUSIONS: The largest group of patients who present with the syndrome of lung hemorrhage and **nephritis** have ANCAs and not **anti-GMB** antibodies. Appropriate tests for antibodies to proteinase 3, antibodies to myeloperoxidase, and anti-GBM antibodies provide reliable guides for making a diagnosis in patients with this pulmonary renal syndrome.
CT Check Tags: Human
*Autoantibodies: BL, blood
Basement Membrane: IM, immunology
*Biological Markers: BL, blood
*Hemorrhage: IM, immunology
*Kidney Glomerulus: IM, immunology
*Lung Diseases: IM, immunology
***Nephritis: IM, immunology**
Predictive Value of Tests
Syndrome
CN 0 (Antibodies, Antineutrophil Cytoplasmic); 0 (Autoantibodies); 0 (Biological Markers)

L2 ANSWER 19 OF 23 MEDLINE
AN 93165992 MEDLINE
DN 93165992
TI [An unusual chronic microvasculitis: Goodpasture's syndrome with late myocardial involvement].
Una insolita microangioite a decorso protratto: sindrome di Goodpasture estesa successivamente al miocardio.
AU Mori R; Corvaglia A G; Frustaci A
CS Istituto di Clinica medica, Universit`a Cattolica del Sacro Cuore, Roma.
SO RECENTI PROGRESSI IN MEDICINA, (1992 Nov) 83 (11) 649-51.
Journal code: R1T. ISSN: 0034-1193.
CY Italy
DT Journal; Article; (JOURNAL ARTICLE)
LA Italian
EM 199305
AB We describe a disease, started in a female young adult patient as an apparent pulmonary siderosis, followed nine years later by an extracapillary proliferative **nephritis**, which developed to uremia in a few months. Later an intra-myocardial vasculitis, responsible of heart failure, appeared. Immune-histochemistry and serological tests exclude a disease mediated by **anti-GMB** antibodies, and pathologic features suggest a vasculitis mainly affecting lungs and kidneys.
CT Check Tags: Case Report; Female; Human
Adult
*Coronary Vessels

AU Pagsberg K; Pedersen G; Hansen F M
SO UGESKRIFT FOR LAEGER, (1989 Aug 21) 151 (34) 2141-4.
Journal code: WM8. ISSN: 0041-5782.
CY Denmark
DT Journal; Article; (JOURNAL ARTICLE)
LA Danish
EM 198912
AB A review is presented of antiglomerular basal membrane antibody-mediated glomerulonephritis (anti-GBM-Ab-**nephritis**) which constitutes 2-5% of all cases of acute glomerulonephritis. The disease frequently commences in the age group 20-30 years but may be encountered in all age groups, in women particularly at 60 years of age. The disease is due to autoantibodies (IgG) to the basal membranes in the glomeruli and alveoli. Deposition of IgG with C3 precipitates an inflammatory reaction which causes renal and possibly also pulmonary damage. It is possible to demonstrate **anti-GMB**-antibodies in the blood and, by means of immunofluorescence microscopy, these and C3 may be demonstrated in the basal membranes in the glomeruli and alveoli. The disease is still serious but introduction of immune-suppressive treatment and plasmapheresis has improved the prognosis considerably.
CT Check Tags: Case Report; Female; Human; Male
Aged
*Autoantibodies: AN, analysis
Complement 3: AN, analysis
English Abstract
*Goodpasture Syndrome: IM, immunology
IgG: AN, analysis
Kidney Glomerulus: IM, immunology
Middle Age
Pulmonary Alveoli: IM, immunology
CN 0 (Autoantibodies); 0 (Complement 3)

L2 ANSWER 21 OF 23 MEDLINE
AN 84293231 MEDLINE
DN 84293231
TI Antinephritic effect of MD-805 [(2R, 4R) -4-methyl-[N2-(3-methyl-1,2,3,4-tetrahydro-8-quinolinesulfonyl) -L-arginyl] -2-piperidine-carboxylic acid monohydrate], a new antithrombin agent, on crescentic-type **anti-GMB nephritis** in rats.
AU Suzuki Y; Yamada H; Ito M
SO NIPPON JINZO GAKKAI SHI. JAPANESE JOURNAL OF NEPHROLOGY, (1984 Apr) 26 (4) 463-73.
Journal code: KMK. ISSN: 0385-2385.
CY Japan
DT Journal; Article; (JOURNAL ARTICLE)
LA Japanese
EM 198412
CT Check Tags: Animal; Comparative Study; Male
Basement Membrane: IM, immunology
Blood Urea Nitrogen
English Abstract
*Glomerulonephritis: DT, drug therapy
Glomerulonephritis: PA, pathology
Heparin: TU, therapeutic use
Kidney Glomerulus: IM, immunology
*Pipecolic Acids: TU, therapeutic use
Rats
Rats, Inbred Strains
Thrombin: AI, antagonists & inhibitors
Urinary Plasminogen Activator: TU, therapeutic use
RN 74863-84-6 (Argatroban); 9005-49-6 (Heparin)
CN EC 3.4.21.5 (Thrombin); EC 3.4.21.73 (Urinary Plasminogen Activator); 0 (Pipecolic Acids)

L2 ANSWER 22 OF 23 MEDLINE
AN 84033172 MEDLINE
DN 84033172
TI Glomerular prostaglandin and thromboxane synthesis in rat nephrotoxic serum **nephritis**. Effects on renal hemodynamics.
AU Lianos E A; Andres G A; Dunn M J
NC AM 06634-02 (NIADDK)
HL 22563 (NHLBI)

synthesis correlated with the presacrifice 24-h protein excretion. 60 min after intravenous infusion of **anti-GMB** serum, glomerular filtration rate (GFR) decreased (0.66 +/- 0.04 to 0.44 +/- 0.03 ml/min per 100 g, P less than 0.05), without a significant change in renal plasma flow (RPF): 1.97 +/- 0.23 to 1.80 +/- 0.23 ml/min per 100 g) and without a change in glomerular PG synthetic rates. At 2 h, GFR and RPF reached a nadir (0.25 +/- 0.04 and 1.3 +/- 0.1 ml/min per 100 g, respectively) coinciding with a fivefold increment in glomerular TxB2. By 3 h GFR and RPF partially recovered to 0.43 +/- 0.07 and 1.77 +/- 0.20 ml/min per 100 g, respectively, P less than 0.05, despite further increments in TxB2 synthesis. This recovery of GFR and RPF coincided with increments in vasodilatory PG, (PGE2 and PGI2). The thromboxane synthetase inhibitor OKY-1581 markedly inhibited platelet and glomerular TxB2 synthesis and preserved GFR at 1, 2, and 3 h. Another thromboxane synthetase inhibitor, UK-38485, also completely inhibited platelet and glomerular TxB2 synthesis and prevented decrements of GFR at 2 and 3 h. A cyclooxygenase inhibitor, ibuprofen, inhibited platelet TxB2 and PGE2 synthesis and significantly reduced glomerular PGE2 but not TxB2 synthesis. In the ibuprofen-treated rats, the partial recoveries of GFR and RPF at 3 h were attenuated. The in vitro glomerular TxB2 synthesis correlated inversely with the presacrifice GFR and filtration fraction. These observations indicate that in anti-GBM **nephritis** there is enhanced synthesis of TxA2 and PG in the glomerulus that mediate changes in renal hemodynamics.

CT Check Tags: Animal; Male; Support, U.S. Gov't, P.H.S.
Blood: PH, physiology
Blood Physiology
Glomerular Filtration Rate: DE, drug effects
Glomerulonephritis: PA, pathology
*Glomerulonephritis: PP, physiopathology
Ibuprofen: AD, administration & dosage
Kidney Glomerulus: AN, analysis
Kidney Glomerulus: PA, pathology
Kidney Glomerulus: PP, physiopathology
Methacrylates: AD, administration & dosage
Nephrotic Syndrome: PA, pathology
*Nephrotic Syndrome: PP, physiopathology
Prostaglandin Antagonists: AD, administration & dosage
*Prostaglandins: BI, biosynthesis
Prostaglandins: PH, physiology
Rabbits
Rats
Rats, Inbred Strains
Renal Circulation
*Thromboxane B2: BI, biosynthesis
Thromboxane B2: PH, physiology
Thromboxane-A Synthase: AI, antagonists & inhibitors
*Thromboxanes: BI, biosynthesis

RN 15687-27-1 (Ibuprofen); 54397-85-2 (Thromboxane B2); 75987-08-5 (OKY 1581)

CN EC 5.3.99.5 (Thromboxane-A Synthase); 0 (Methacrylates); 0 (Prostaglandin Antagonists); 0 (Prostaglandins); 0 (Thromboxanes)

L2 ANSWER 23 OF 23 MEDLINE

AN 79155018 MEDLINE

DN 79155018

TI Radioimmunologic method for detection of antitubular basement membrane antibodies.

AU Graindorge P P; Mahieu P R

SO KIDNEY INTERNATIONAL, (1978 Dec) 14 (6) 594-606.
Journal code: KVB. ISSN: 0085-2538.

CY GERMANY, WEST: Germany, Federal Republic of

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 197908

AB A radioimmunoassay for detection of antitubular basement membrane (TBM) antibodies was set up using a human TBM antigen (mol wt, 70,000 daltons), purified after collagenase treatment of the insoluble membrane by preparative polyacrylamide electrophoresis, and labeled with iodine 125. Free labeled antigens were separated from those bound to immunoglobulins by a 20% polyethylene glycol (mol wt, 6,000 daltons) solution. In the presence of normal human or Brown Norway rat sera, less than 10% of the

antibodies were directed against the noncollagenous polypeptides of TBM but that the anti-GBM antibodies mainly reacted with the collagenous polypeptides of TBM and GBM. Finally, it was found that the sera of 2 patients out of 15 presenting with lupus **nephritis** contained a significant anti-TBM-binding activity, mainly directed against the noncollagenous material of TBM.

CT Check Tags: Animal
Amino Acids: AN, analysis
Antigens: AN, analysis
*Autoantibodies: AN, analysis
Basement Membrane: IM, immunology
Carbohydrates: AN, analysis
*Kidney Diseases: IM, immunology
Kidney Glomerulus: IM, immunology
*Kidney Tubules: IM, immunology
*Radioimmunoassay: MT, methods
Rats

=> d his

(FILE 'HOME' ENTERED AT 10:32:01 ON 30 MAR 2001)

FILE 'BIOSIS, CAPLUS, EMBASE, CANCERLIT, MEDLINE' ENTERED AT 10:32:43 ON 30 MAR 2001

L1 47 S ANTI-GMB
L2 23 S L1 AND NEPHRITIS

=> s l2 and (alpha2u globulin)

L3 0 L2 AND (ALPHA2U GLOBULIN)

=> s l2 and (major urinary protein0

UNMATCHED LEFT PARENTHESIS 'AND (MAJOR'
The number of right parentheses in a query must be equal to the number of left parentheses.

=> s l2 and (major urinary protein)

4 FILES SEARCHED...
L4 0 L2 AND (MAJOR URINARY PROTEIN)

=> s l1 and (major urinary protein)

4 FILES SEARCHED...
L5 0 L1 AND (MAJOR URINARY PROTEIN)

=> s (mouse glomular basal membrane0

UNMATCHED LEFT PARENTHESIS '(MOUSE'
The number of right parentheses in a query must be equal to the number of left parentheses.

=> s (mouse glomular basal membrane)

L6 0 (MOUSE GLOMULAR BASAL MEMBRANE)

=> s nagai/au

L7 2 NAGAI/AU

=> d l7 1-2 all

L7 ANSWER 1 OF 2 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 1998002275 EMBASE
TI Laparoscopic-assisted colectomy for advanced colorectal carcinomas - Feasibility of lymph node dissection.
AU Konishi F.; **Nagai**; Okada M.; Ozawa A.; Kanazawa K.
CS F. Konishi, Department of Surgery, Jichi Medical School, Tochigi, Japan

invasive carcinomas in open laparotomy. Laparoscopic-assisted colectomy and lymphnode dissection have been done in 29 cases of advanced colorectal carcinoma. In this study, the technical aspect of lymphnode dissection in the laparoscopic procedure was presented, and it was considered that this procedure is a curative surgery for advanced colorectal carcinoma, provided that the surgeon is technically well experienced and the patient is properly selected.

CT Medical Descriptors:
*colorectal cancer: ET, etiology
*colorectal cancer: SU, surgery
*lymph node metastasis: CO, complication
*lymph node metastasis: SU, surgery
colon resection
lymph node dissection
laparoscopic surgery
surgical technique
human
male
female
major clinical study
aged
adult
article

L7 ANSWER 2 OF 2 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 75026521 EMBASE
DN 1975026521
TI A new pyroglutamylpeptide (Pyr Lys Ser) isolated from the venom of Agkistrodon halys blomhoffii.
AU Okada K.; **Nagai**; Kato H.
CS Fac. Pharmaceut. Sci., Univ. Kanazawa, Japan
SO Experientia, (1974) 30/5 (459-460).
CODEN: EXPEAM
DT Journal
FS 037 Drug Literature Index
029 Clinical Biochemistry
030 Pharmacology
LA English
CT Medical Descriptors:
*cyclopentanophosphatidyl n,n dimethylethanolamine
*drug analysis
*hydrolysis
*ruvalcaba syndrome
theoretical study
Drug Descriptors:
*bradykinin
*venom
RN (bradykinin) 58-82-2, 5979-11-3

=> s gBM

L8 7090 GBM

=> s l8 and nephrit?

L9 1881 L8 AND NEPHRIT?

=> s l9 and anti

L10 1404 L9 AND ANTI

=> s l10 and (alpha 2u globulin)\

MISSING OPERATOR GLOBULIN)\
The search profile that was entered contains terms or
nested terms that are not separated by a logical operator.

=> s l10 and (alpha globulin)

L11 0 L10 AND (ALPHA GLOBULIN)

=> s l14 and FABP

L16 0 L14 AND FABP

=> s l14 and antibod?

L17 58 L14 AND ANTIBOD?

=> d l17 1-58 all

L17 ANSWER 1 OF 58 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1998:87129 BIOSIS
DN PREV199800087129
TI Influence of endotoxin contamination on **anti-GBM** **antibody** induced glomerular injury in rats: Technical note.
AU Karkar, Ayman M. (1); Rees, Andrew J.
CS (1) Renal Unit, Dep. Med., Royal Postgrad. Medical Sch., Hammersmith Hosp., Du Cane Road, London W12 0NN UK
SO Kidney International, (Dec., 1997) Vol. 52, No. 6, pp. 1579-1583. ISSN: 0085-2538.
DT Article
LA English
AB It is accepted that the main determinant of glomerular injury in experimental nephrotoxic **nephritis** is the administered dose of **anti**-glomerular basement membrane (**GBM**) **antibody** . However, there are other factors that can enhance the severity of such injury including small doses of bacterial lipopolysaccharide (LPS). In the present study, we have assessed whether preparations of **anti-GBM antibody** contaminated with different concentrations of endotoxin could influence the severity of glomerular injury in the heterologous phase of nephrotoxic **nephritis**. We have also examined the efficacy of different laboratory methods to isolate an endotoxin-free **anti-GBM antibody**, and to purify **anti-GBM antibody** preparations from endotoxin. Preparations of **anti-GBM antibody** (nephrotoxic **globulin**) isolated from nephrotoxic serum by the sodium sulphate precipitation method contained variable concentrations of endotoxin. Administration of these preparations in equal doses into clean rats, which had no established acute phase response, markedly aggravated the severity of glomerular injury. However, preparations contained less than 50 pg/ml of endotoxin appeared to have no significant effect on such injury. Furthermore, isolation of **anti-GBM** **antibody** from nephrotoxic serum by affinity chromatography, using Staphylococcus **protein**-A column, proved to be a reliable method not only for the isolation of an IgG (nephrotoxic **antibody**) free from other serum contaminants, but also for purification of endotoxin contaminated preparations of **anti-GBM antibody** . These observations have practical implications in studying models of **nephritis** as our results show that the glomerular injury, which is usually considered to be a sole function of the mass of **antibody** bound to **GBM**, is profoundly influenced by minor endotoxin contamination of the **anti-GBM antibody**.
CC Urinary System and External Secretions - Pathology *15506
Toxicology - General; Methods and Experimental *22501
Biochemical Studies - Proteins, Peptides and Amino Acids *10064
Biochemical Studies - Carbohydrates *10068
BC Hominidae 86215
IT Major Concepts
Urinary System (Chemical Coordination and Homeostasis)
IT Diseases
nephrotoxic **nephritis**: urologic disease
IT Chemicals & Biochemicals
anti-glomerular basement membrane **antibody**;
bacterial lipopolysaccharide; endotoxin: contamination, influence
ORGN Super Taxa
Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
human (Hominidae): patient
ORGN Organism Superterms
Animals; Chordates; Humans; Mammals; Primates; Vertebrates

cholesterol content in plasma was lower than that of the **nephritic** control rats. Histological observation demonstrated that this agent suppressed the incidence of crescent formation, adhesion of capillary wall to Bowman's capsule and fibrinoid necrosis in the glomeruli. Furthermore, butein suppressed the accumulation of leukocytes, including CD4-positive cells and CD8-positive cells in the glomeruli. However, butein failed to suppress the production of the **antibody** against rabbit gamma-**globulin** and the deposition of rat-IgG on the **GBM**. These results suggest that butein may be a useful medicine against rapidly progressive glomerulonephritis, which is characterized by severe glomerular lesions with diffuse crescents.

CC Biochemical Studies - General 10060
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Biochemical Studies - Sterols and Steroids 10067
Biophysics - Membrane Phenomena *10508
Pathology, General and Miscellaneous - Necrosis *12510
Blood, Blood-Forming Organs and Body Fluids - Blood Cell Studies *15004
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System *15008
Urinary System and External Secretions - Pathology *15506
Pharmacology - Immunological Processes and Allergy *22018
Immunology and Immunochemistry - General; Methods *34502
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508

BC Muridae *86375

IT Major Concepts
Blood and Lymphatics (Transport and Circulation); Immune System (Chemical Coordination and Homeostasis); Membranes (Cell Biology); Pathology; Pharmacology; Urinary System (Chemical Coordination and Homeostasis)

IT Chemicals & Biochemicals
BUTEIN; CHOLESTEROL

IT Miscellaneous Descriptors
ADHESIONS; BUTEIN; CHOLESTEROL; FIBROID NECROSIS; IMMUNOGLOBULIN G; IMMUNOSUPPRESSANT-DRUG; LEUKOCYTES; **PROTEIN** EXCRETION

ORGN Super Taxa
Muridae: Rodentia, Mammalia, Vertebrata, Chordata, Animalia

ORGN Organism Name
Muridae (Muridae)

ORGN Organism Superterms
animals; chordates; mammals; nonhuman vertebrates; nonhuman mammals; rodents; vertebrates

RN 487-52-5 (BUTEIN)
57-88-5 (CHOLESTEROL)

L17 ANSWER 3 OF 58 BIOSIS COPYRIGHT 2001 BIOSIS

AN 1994:537474 BIOSIS

DN PREV199497550474

TI Acteoside, a component of Stachys sieboldii MIQ, may be a promising antinephritic agent (2): Effect of acteoside on leukocyte accumulation in the glomeruli of **nephritic** rats.

AU Hayashi, Kazumi; Nagamatsu, Tadashi; Ito, Mikio; Hattori, Tomohisa; Suzuki, Yoshio

CS Dep. Pharmacol., Fac. Pharmacy, Meijo Univ., 150 Yagotoyama, Tenpaku-ku, Nagoya 468 Japan

SO Japanese Journal of Pharmacology, (1994) Vol. 66, No. 1, pp. 47-52. ISSN: 0021-5198.

DT Article

LA English

AB We investigated the effect of acteoside in comparison with that of cyclosporin A on leukocyte accumulation in the glomeruli of rats with crescentic-type **anti**-glomerular basement membrane (**GBM**) **nephritis**. Acteoside given p.o. at a dose of 30 mg/kg once a day for 15 consecutive days after treatment with **anti-GBM** serum markedly suppressed the urinary **protein** as well as glomerular histological changes. Acteoside given p.o. for 5 or 15 consecutive days markedly suppressed the accumulation of total leukocytes, ED-1-positive cells (monocytes/macrophages), CD4-positive cells, CD8-positive cells, interleukin-2-receptor-positive cells (activated T cells) and Ia-positive cells in the glomeruli. These effects of cyclosporin A (20 mg/kg/day, p.o.) were also as potent as those of acteoside (30 mg/kg/day, p.o.). Cyclosporin A also strongly suppressed the

IT *Major Concepts
Biochemistry and Molecular Biophysics; Pathology; Pharmacognosy (Pharmacology); Pharmacology; Urinary System (Chemical Coordination and Homeostasis)
IT Chemicals & Biochemicals
ACTEOSIDE
IT Miscellaneous Descriptors
ACETOSIDE; ANTIINFLAMMATORY-DRUG; DIURETIC-DRUG; EFFICACY; PHARMACEUTICAL BOTANY; PHARMACODYNAMICS
ORGN Super Taxa
Labiatae: Dicotyledones, Angiospermae, Spermatophyta, Plantae; Malvaceae: Dicotyledones, Angiospermae, Spermatophyta, Plantae; Muridae: Rodentia, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
Malvaceae (Malvaceae); Muridae (Muridae); Stachys sieboldii (Labiatae)
ORGN Organism Superterms
angiosperms; animals; chordates; dicots; mammals; nonhuman mammals; nonhuman vertebrates; plants; rodents; spermatophytes; vascular plants; vertebrates
RN 61276-17-3 (ACTEOSIDE)

L17 ANSWER 4 OF 58 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1994:396358 BIOSIS
DN PREV199497409358
TI Acteoside, a component of Stachys sieboldii MIQ, may be a promising antinephritic agent: Effect of acteoside on crescentic-type **anti -GBM nephritis** in rats.
AU Hayashi, Kazumi; Nagamatsu, Tadashi; Ito, Mikio; Hattori, Tomohisa; Suzuki, Yoshio
CS Dep. Pharmacol., Fac. Pharmacy, Meijo Univ., 150 Yagotoyama, Tenpaku-ku, Nagoya 468 Japan
SO Japanese Journal of Pharmacology, (1994) Vol. 65, No. 2, pp. 143-151. ISSN: 0021-5198.
DT Article
LA English
AB Effects of acteoside (ACT) on crescentic-type **anti-GBM nephritis** in rats were investigated. When rats were treated with ACT from the 1st day after i.v. injection of **anti-GBM** serum, ACT inhibited the elevation of **protein** excretion into urine. In the ACT-treated rats, cholesterol and creatinine contents and **antibody** production against rabbit gamma-**globulin** in the plasmas were lower than those of the **nephritic** control rats. Histological observation demonstrated that this agent suppressed hypercellularity and the incidence of crescent formation, adhesion of capillary wall to Bowman's capsule and fibrinoid necrosis in the glomeruli. Furthermore, rat-IgG and C-3 deposits on the **GBM** were significantly less in the ACT-treated group than in the control **nephritic** group. When the treatment was started from the 20th day after i.v. injection of **anti-GBM** serum, by which the disease had been established, ACT resulted in a similar effect on the **nephritic** rats as stated above. These results suggest that ACT may be a useful medicine against rapidly progressive glomerulonephritis, which is characterized by severe glomerular lesions with diffuse crescents.
CC Cytology and Cytochemistry - Animal *02506
Biochemical Studies - General 10060
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Pathology, General and Miscellaneous - Inflammation and Inflammatory Disease *12508
Pathology, General and Miscellaneous - Therapy 12512
Urinary System and External Secretions - Pathology *15506
Pharmacology - Sense Organs, Associated Structures and Functions *22031
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
BC Labiatae 26230
Muridae *86375
IT Major Concepts
Cell Biology; Immune System (Chemical Coordination and Homeostasis); Pathology; Pharmacology; Urinary System (Chemical Coordination and Homeostasis)
IT Chemicals & Biochemicals
ACTEOSIDE
IT Miscellaneous Descriptors
ACTEOSIDE; HYPERCELLULARITY SUPPRESSION; IMMUNOGLOBULIN G

anti-GBM nephritis in rats.

AU Hattori, Tomohisa; Furuta, Kazuya; Hayashi, Kazumi; Nagamatsu, Tadashi; Ito, Mikio; Suzuki, Yoshio

CS Dep. Pharmacol., Fac. Pharm., Meijo Univ., 150 Yagotoyama, Tenpaku-ku, Nagoya 468 Japan

SO Japanese Journal of Pharmacology, (1992) Vol. 60, No. 3, pp. 187-195. ISSN: 0021-5198.

DT Article

LA English

AB Effects of phellodendrine (OB-5) on crescentic-type **anti-GBM nephritis** in rats and the cell number of the various leukocyte subpopulations in the glomeruli of the **nephritic** rats were investigated. OB-5 at 25, 50 and 100 mg/kg/day, p.o. prevented the urinary **protein** excretion by the 19th day after i.v.-injection of **anti-GBM** serum. In the OB-5-treated rats, plasma cholesterol and creatinine contents were lower than those of the control rats throughout the 40-day experimental period. Histopathological observations demonstrated that OB-5 inhibited the incidence of crescent formation, adhesion and fibrinoid necrosis in the glomeruli by the 41st day. OB-5 did not affect the plasma **antibody** titer against rabbit gamma **globulin**. The increases in total leukocytes, macrophages, cytotoxic/suppressor T cells, Ia positive cells, and IL- 2 receptor positive cells in the glomeruli in OB-5, 100 mg/kg-treated rats as well as those of the animals treated with azathioprine or cyclosporin A were lower than those of the **anti-GBM nephritic** control. These results indicate that OB-5 was effective in crescentic-type **anti-GBM nephritis** and the antinephritic mechanisms of this agent may be due to its ability to inhibit the proliferation or the migration of macrophages and cytotoxic T lymphocytes in the glomeruli.

CC Cytology and Cytochemistry - Animal *02506
Physical Anthropology; Ethnobiology *05000
Biochemical Studies - General 10060
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Biochemical Studies - Carbohydrates 10068
Pathology, General and Miscellaneous - Inflammation and Inflammatory Disease *12508
Blood, Blood-Forming Organs and Body Fluids - Blood Cell Studies *15004
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System *15008
Urinary System and External Secretions - Pathology *15506
Endocrine System - General *17002
Pharmacology - Immunological Processes and Allergy *22018
Pharmacology - Urinary System *22032
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
Plant Physiology, Biochemistry and Biophysics - Chemical Constituents *51522
Pharmacognosy and Pharmaceutical Botany *54000

BC Rutaceae 26685
Muridae *86375

IT Major Concepts
Anthropology; Biochemistry and Molecular Biophysics; Blood and Lymphatics (Transport and Circulation); Cell Biology; Endocrine System (Chemical Coordination and Homeostasis); Immune System (Chemical Coordination and Homeostasis); Pathology; Pharmacognosy (Pharmacology); Pharmacology; Urinary System (Chemical Coordination and Homeostasis)

IT Miscellaneous Descriptors
ANTI-GLOMERULAR BASEMENT MEMBRANE **NEPHRITIS**; CELL MIGRATION; CELL PROLIFERATION; IMMUNOLOGIC-DRUG; INTERLEUKIN-2 RECEPTOR; JAPANESE TRADITIONAL MEDICINE; MACROPHAGE; PHARMACODYNAMICS; RENAL-ACTING-DRUG; T LYMPHOCYTE

ORGN Super Taxa
Muridae: Rodentia, Mammalia, Vertebrata, Chordata, Animalia; Rutaceae: Dicotyledones, Angiospermae, Spermatophyta, Plantae

ORGN Organism Name
Muridae (Muridae); Phellodendron amurense (Rutaceae)

ORGN Organism Superterms
angiosperms; animals; chordates; dicots; mammals; nonhuman mammals; nonhuman vertebrates; plants; rodents; spermatophytes; vascular plants; vertebrates

0.5 mg/kg/day) and TEI-6122 (0.25 or 0.5 mg/kg/day) significantly reduced urinary **protein** by 30 to 50% of that of the control at the late stage of **nephritis**. These test compounds also suppressed the increase of blood urea nitrogen and the development of alteration in the glomeruli by the 40th day. Both TEI-5178 (0.5 mg/kg/day) and TEI-6122 (0.5 mg/kg/day) significantly suppressed the production of **antibody** to rabbit .gamma.-**globulin** in nepritic rats. This was not the case with PGE1, however. In additional experiments to clarify the antinephritic mechanisms of the test compounds, it was found that 15 min after one subcutaneous injection of PGE1 (1.0 mg/kg), TEl-5178 (0.5 mg/kg) or TEI-6122 (0.5 mg/kg), systolic blood pressure in the **nephritic** rats was transiently reduced by 50 to 60%. On the other hand, these test compounds augmented renal blood flow (20-50%) from 45 min after the injection. The relationship between the antinephritic effect and these subsequent findings will be discussed.

CC Biochemical Studies - Lipids 10066
Pathology, General and Miscellaneous - Inflammation and Inflammatory Disease 12508
Pathology, General and Miscellaneous - Therapy 12512
Cardiovascular System - Blood Vessel Pathology *14508
Urinary System and External Secretions - Pathology *15506
Endocrine System - General *17002
Pharmacology - Endocrine System *22016
Pharmacology - Urinary System *22032
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508

BC Muridae 86375

IT Miscellaneous Descriptors
PROSTAGLANDIN E-1 THIAPROSTAGLANDIN E-1 HORMONE-DRUG RENAL-ACTING-DRUG **ANTI**-GLOMERULAR BASEMENT MEMBRANE **NEPHRITIS** BLOOD PRESSURE RENAL BLOOD FLOW

RN 745-65-3 (PROSTAGLANDIN E-1)
83009-96-5 (TEI-5178)
83058-69-9 (TEI-6122)

L17 ANSWER 7 OF 58 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1984:218795 BIOSIS
DN BA77:51779
TI FACTORS AFFECTING SEVERITY OF INJURY DURING NEPHRO TOXIC **NEPHRITIS** IN RABBITS.
AU VAN ZYL SMIT R; REES A J; PETERS D K
CS DEP. MED., ROYAL POSTGRADUATE MED. SCH., HAMMERSMITH HOSP., DUCANE ROAD, LONDON W12 0H5, UK.
SO CLIN EXP IMMUNOL, (1983) 54 (2), 366-372.
CODEN: CEXIAL. ISSN: 0009-9104.
FS BA; OLD
LA English
AB All 22 rabbits injected with sheep **globulin** containing high titers of **antibodies** to rabbit glomerular basement membrane (**GBM**)-nephrotoxic globulin (**NTG**)-developed antibodies to **sheep** IgG. Despite this only 15 rabbits developed obvious autologous phase injury. Eleven days after injection of NTG titers of autologous antibody to **sheep** IgG were similar in rabbits with and without definite autologous phase injury but were detected earlier and rose significantly more rapidly in those with autologous phase injury. In experiments on heterologous phase injury after i.v. injection of NTG, binding of defined amounts of nephrotoxic antibodies (NTAb) **to** the GBM after **bolus** injection caused significantly more injury, assessed by proteinuria, than **binding** of similar amounts of NTAb after infusion of NTG over 3 h ($P < 0.02$ Student's paired t-test). In in vitro experiments, aliquots of homogenized rabbit kidney taken 2 days after injection of NTG bound appreciable amounts of rabbit anti-sheep **Ig**, whereas homogenates of kidneys taken 20 days after NTG showed no such binding. Evidently the rate of deposition of NTAb in kidney influences the severity of injury in heterologous and autologous phases of NTN (nephrotoxic nephritis), and **antigenic** sites or heterologous IgG fixed to the GBM become **saturated** during the autologous phase of injury.

CC Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Biochemical Studies - Carbohydrates 10068
Biophysics - Molecular Properties and Macromolecules 10506
Biophysics - Membrane Phenomena 10508
Pathology, General and Miscellaneous - Inflammation and Inflam-

Leporidae 86040
IT Miscellaneous Descriptors
SHEEP **GLOBULIN** GLOMERULAR BASEMENT MEMBRANE NEPHRO TOXIC **GLOBULIN** HOMOGENIZED KIDNEY IMMUNO **GLOBULIN** G **ANTIBODIES PROTEINURIA**

L17 ANSWER 8 OF 58 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1984:202409 BIOSIS
DN BA77:35393
TI **ANTI** GLOMERULAR BASEMENT MEMBRANE AUTO **ANTIBODIES** IN THE BROWN NORWAY RAT DETECTION BY A SOLID PHASE RADIO IMMUNOASSAY.
AU BOWMAN C; PETERS D K; LOCKWOOD C M
CS RENAL UNIT, DEP. MED., ROYAL POSTGRADUATE MED. SCH., HAMMERSMITH HOSP., DU CANE ROAD, LONDON W12 0HS, UK.
SO J IMMUNOL METHODS, (1983) 61 (3), 325-334.
CODEN: JIMMBG. ISSN: 0022-1759.
FS BA; OLD
LA English
AB A solid-phase radioimmunoassay (RIA) is described for the detection of IgG autoantibodies to glomerular basement membrane (**GBM**) induced in the Brown Norway rat by mercuric chloride. The assay involves the adsorption of a collagenase digest of **GBM** to plastic microtiter plates and detection of bound **antibody** with affinity purified radiolabeled rabbit **anti**-rat IgG. Comparison with existing immunofluorescence methods for detection of **anti-GBM antibody** showed that the solid-phase RIA is highly sensitive, allowing detection of **antibody** in solutions with as low as 0.5 ng **protein**/ml. The assay is suitable for detection of **anti-GBM antibody** both in serum and in eluates from **nephritic** kidneys. The assay was specific in competitive studies of inhibition brought about by **GBM**, keyhole limpet antigen and ovalbumin. This solid-phase RIA is reproducible, robust and easy to perform.
CC Radiation - Radiation and Isotope Techniques 06504
Ecology; Environmental Biology - Water Research and Fishery Biology 07517
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Biochemical Studies - Carbohydrates 10068
Biochemical Studies - Minerals 10069
Enzymes - Methods 10804
Pathology, General and Miscellaneous - Inflammation and Inflammatory Disease 12508
Metabolism - Carbohydrates *13004
Metabolism - Proteins, Peptides and Amino Acids *13012
Blood, Blood-Forming Organs and Body Fluids - Blood and Lymph Studies 15002
Bones, Joints, Fasciae, Connective and Adipose Tissue - Pathology *18006
Toxicology - General; Methods and Experimental 22501
Immunology and Immunochemistry - General; Methods *34502
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
Invertebrata, Comparative and Experimental Morphology, Physiology and Pathology - Mollusca 64026
BC Gastropoda 61200
Leporidae 86040
Muridae 86375
IT Miscellaneous Descriptors
NEPHRITIC KIDNEY COLLAGENASE DIGEST RABBIT **ANTI** RAT **ANTIBODY** IMMUNO **GLOBULIN** G KEYHOLE LIMPET HEMO CYANIN OV ALBUMIN MERCURIC CHLORIDE
RN 7487-94-7 (MERCURIC CHLORIDE)
9001-12-1 (COLLAGENASE)

L17 ANSWER 9 OF 58 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1983:334927 BIOSIS
DN BA76:92419
TI **ANTI** GLOMERULAR BASEMENT MEMBRANE **ANTIBODY** **ANTIBODY** SPECIFICITY IN DIFFERENT FORMS OF GLOMERULO **NEPHRITIS**.
AU WIESLANDER J; BYGREN P; HEINEGARD D
CS DEP. NEPHROL., UNIV. HOSP. LUND, S-221 85 LUND, SWEDEN.
SO KIDNEY INT, (1983) 23 (6), 855-861.

collagenase digestion. Pepsin digestation destroyed the antigen(s). The **antibodies** were of a different class, i.e., the patients with systemic lupus erythematous had IgG and IgA as well as IgM **antibodies**; the patients with periarteritis nodosa had IgM or IgG and IgA **antibodies**, while the patients with IgA-related **nephritis** had the highest recorded titers of IgA but also had IgG as well as IgM **antibodies**. None of the patients had **antibodies** directed against triple helical collagen. The **antibody** response in **anti-GBM antibody** -related **nephritis** is different both with respect to antigen and **antibody** class and depends on the underlying disease syndrome.

CC Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Biochemical Studies - Porphyrins and Bile Pigments 10065
Biophysics - General Biophysical Techniques 10504
Enzymes - Methods *10804
Movement 12100
Pathology, General and Miscellaneous - Inflammation and Inflammatory Disease 12508
Metabolism - Carbohydrates *13004
Metabolism - Proteins, Peptides and Amino Acids *13012
Urinary System and External Secretions - General; Methods *15501
Urinary System and External Secretions - Pathology *15506
Bones, Joints, Fasciae, Connective and Adipose Tissue - General; Methods 18001
Bones, Joints, Fasciae, Connective and Adipose Tissue - Pathology *18006
Integumentary System - Pathology *18506
Immunology and Immunochemistry - General; Methods 34502
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508

BC Hominidae 86215

IT Miscellaneous Descriptors
HUMAN IMMUNO **GLOBULINS** COLLAGENASE PEPSIN DIGESTION GUANIDINE HYDRO CHLORIDE ENZYME LINKED IMMUNO SORBENT ASSAY SERA GOODPASTURE SYNDROME LUPUS ERYTHEMATOSUS PERI ARTERITIS NODOSA

RN 9001-12-1 (COLLAGENASE)
9001-75-6 (PEPSIN)
50-01-1Q, 106946-18-3Q (GUANIDINE HYDRO CHLORIDE)

L17 ANSWER 10 OF 58 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1982:237810 BIOSIS
DN BA74:10290
TI QUANTITATIVE STUDIES OF IN-SITU IMMUNE COMPLEX GLOMERULO **NEPHRITIS** IN THE RAT INDUCED BY PLANTED CATIONIZED ANTIGEN.
AU OITE T; BATSFORD S R; MIHATSCH M J; TAKAMIYA H; VOGT A
CS INST. IMMUNOL., ZENTRUM HYGIENE UNIV. FREIBURG, 7800 FREIBURG IM BREISGAU, FRG.
SO J EXP MED, (1982) 155 (2), 460-474.
CODEN: JEMEAV. ISSN: 0022-1007.
FS BA; OLD
LA English
AB Cationized human IgG can bind to the rat glomerular basement membrane (**GBM**), act as planted antigen and induce in situ immune complex formation accompanied by severe glomerulonephritis. Perfusion of highly cationized human IgG (isoelectric point > 9.5) via the left renal artery resulted in preferential localization within the perfused kidney (up to 56% of dose injected); after i.v. administration, only 4% was bound to the kidneys. The planted antigen was localized along the glomerular capillary walls and was accessible for **antibody** administered i.v. 1 h after perfusion, when virtually no antigen remained in the circulation. Persistence of cationized human IgG in the perfused kidney was markedly prolonged when complexed with **antibody**; 1/2 the cationized human IgG was still present after 12 days. There was a difference in the disappearance rates of antigen and **antibody**; cationized human IgG was removed faster from the kidney than the **antibody**, the binding of which remained almost unchanged during the 1st wk. Renal perfusion of a minimum of 20 .mu.g of cationized human IgG, followed by i.v. injection of **antibody**, regularly induced severe glomerulonephritis with a **proteinuria** of at least 100 mg/24 h. The degree and the persistence of **proteinuria** induced depended on the dose of cationized human IgG perfused. Experiments using radiolabeled antigen and **antibody** showed that after renal perfusion of 20 .mu.g cationized human IgG, 11.1 .mu.g was kidney bound at

Anatomy and Histology, General and Comparative - Microscopic and Ultramicroscopic Anatomy *11108
Pathology, General and Miscellaneous - Inflammation and Inflammatory Disease 12508
Metabolism - General Metabolism; Metabolic Pathways 13002
Cardiovascular System - General; Methods 14501
Blood, Blood-Forming Organs and Body Fluids - Blood and Lymph Studies 15002
Urinary System and External Secretions - General; Methods 15501
Urinary System and External Secretions - Anatomy 15502
Urinary System and External Secretions - Physiology and Biochemistry 15504
Urinary System and External Secretions - Pathology *15506
Routes of Immunization, Infection and Therapy 22100
Immunology and Immunochemistry - General; Methods 34502
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508

BC Leporidae 86040
Hominidae 86215
Muridae 86375

IT Miscellaneous Descriptors
RABBIT HUMAN IMMUNO **GLOBULIN** G KIDNEY GLOMERULAR BASEMENT MEMBRANE **PROTEINURIA** SUBEPITHELIAL SPACE SLIT PORES

L17 ANSWER 11 OF 58 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1981:278664 BIOSIS
DN BA72:63648
TI IMMUNO ENZYMATIC STUDY OF THE **PROTEIN** PATHWAY THROUGH THE GLOMERULAR BARRIER IN RAT GLOMERULO **NEPHRITIDES**.
AU BARIETY J; BELLON B; SAPIN C; KUHN J; DRUET P; HINGLAIS N; GIRAUD J-P; BELAIR M-F; PAING M; LALIBERTE F
CS CLINIQUE MED., HOPITAL BROUSSAIS, 96, RUE DIDOT, 75674 PARIS, CEDEX 14, FRANCE.
SO KIDNEY INT, (1981) 19 (5), 663-677.
CODEN: KDYIA5. ISSN: 0085-2538.
FS BA; OLD
LA English
AB Circulating antihorseradish peroxidase (HRP) IgG **antibodies** were used in the rat to study the glomerular leakage of **proteins** in glomerulonephritis (GN) induced by aminonucleoside (AN) and in glomerulonephritis induced by mercuric chloride to produce antiglomerular basement membrane (**GBM antibodies**. In ANGN, autologous albumin and fibrinogen were also detected by immunoperoxidase techniques. In both types of GN, the **proteins** studied were observed in the glomerular urinary space and proximal tubular cells. No channels were visible in the lamina densa. No accumulation of **proteins** was seen under the epithelial slits that were not closed. In ANGN, accumulation of **proteins** was observed in the subepithelial space where the podocytes act as a barrier (closed slits, subepithelial blind pockets, areas covered by broad sheets of cytoplasm), but no accumulation was seen in the lamina rara externa under normal or enlarged slits and areas of large epithelial cytoplasm detachment. Statistical analysis showed that in ANGN, at the time of maximal **proteinuria**, the number of micropinocytotic vesicles for the **GBM**-embedded part of podocytes was not increased as compared with controls. Such vesicles were not labeled. Apparently the permeability of the **GBM** is diffusely increased and that the plasma **proteins** pass into the urinary space via and extracellular pathway.
CC Microscopy Techniques - Histology and Histochemistry 01056
Mathematical Biology and Statistical Methods 04500
Biochemical Studies - General 10060
Biochemical Studies - Nucleic Acids, Purines and Pyrimidines 10062
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Biochemical Studies - Carbohydrates 10068
Biophysics - Membrane Phenomena *10508
Enzymes - Methods *10804
Movement 12100
Pathology, General and Miscellaneous - Inflammation and Inflammatory Disease *12508
Metabolism - Proteins, Peptides and Amino Acids *13012
Blood, Blood-Forming Organs and Body Fluids - Blood and Lymph Studies 15002

AN 1981:239368 BIOSIS
DN BA72:24352
TI MASUGI **NEPHRITIS** AN ADDITIONAL USE FOR CROSS REACTIVITY ASSESSMENT OF PLASMA **PROTEINS**.
AU NISHIHARA T; KUSUYAMA Y; SAITO K; TAKENAKA T
CS DEP. PATHOL., WAKAYAMA MED. COLL., WAKAYAMA 640, JPN.
SO WAKAYAMA MED REP, (1980 (RECD 1981)) 23 (3), 89-98.
CODEN: WKMRAH. ISSN: 0511-084X.
FS BA; OLD
LA English
AB The development of Masugi **nephritis**, an experimental **anti**-glomerular basement membrane (**GBM**) disease, is widely recognized to coincide with characteristic linear depositon of Ig and complement components. Renal glomeruli of rat, mouse, hamster and gerbil affected with this disease were used for cross-reactivity assessment of IgG, C3 and fibrinogen among a mammalian species. In immunofluorescent preparations, homologs of IgG and C3 were detected among rat, mouse, hamster and gerbil. **Antibody** to guinea pig IgG and 1 to human IgG could react with gerbil IgG but not with IgG of rat, mouse and hamster. **Anti**-rat fibrinogen was also located along the **GBM** and sometimes at the glomeruli periphery, where fibrin-related antigens were perhaps deposited, in each animal tested. Apparently the use of the renal glomerulus for the assessment of antigenic similarities of certain plasma **proteins** among laboratory animals is of considerable interest.
CC Cytology and Cytochemistry - Animal 02506
Cytology and Cytochemistry - Human 02508
Comparative Biochemistry, General 10010
Biochemical Methods - Proteins, Peptides and Amino Acids 10054
Biochemical Methods - Carbohydrates 10058
Biochemical Studies - Proteins, Peptides and Amino Acids *10064
Biochemical Studies - Carbohydrates 10068
Enzymes - Physiological Studies *10808
Pathology, General and Miscellaneous - Comparative 12503
Pathology, General and Miscellaneous - Inflammation and Inflammatory Disease 12508
Blood, Blood-Forming Organs and Body Fluids - Blood and Lymph Studies *15002
Urinary System and External Secretions - Physiology and Biochemistry 15504
Urinary System and External Secretions - Pathology *15506
Immunology and Immunochemistry - General; Methods *34502
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
BC Hominidae 86215
Caviidae 86300
Cricetidae 86310
Muridae 86375
IT Miscellaneous Descriptors
RAT MOUSE HAMSTER GERBIL GUINEA-PIG HUMAN EXPERIMENTAL **ANTI** GLOMERULAR BASEMENT MEMBRANE DISEASE FIBRINOGEN IMMUNO **GLOBULIN** G COMPLEMENT C-3
RN 56626-15-4 (COMPLEMENT C-3)

L17 ANSWER 13 OF 58 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1981:196910 BIOSIS
DN BA71:66902
TI RECURRENCE OF **ANTI** GLOMERULAR BASEMENT MEMBRANE **ANTIBODY** MEDIATED GLOMERULO **NEPHRITIS** IN AN ISO GRAFT.
AU ALMKUIST R D; BUCKALEW V M JR; HIRSZEL P; MAHER J F; JAMES P M; WILSON C B
CS BOWMAN GRAY SCH. OF MED., WAKE FORREST UNIV., WINSTON-SALEM, N.C. 27103.
SO CLIN IMMUNOL IMMUNOPATHOL, (1981) 18 (1), 54-60.
CODEN: CLIIAT. ISSN: 0090-1229.
FS BA; OLD
LA English
AB A renal isograft was performed without immunosuppression in a patient with Goodpasture's syndrome, whose **anti**-glomerular basement membrane (**GBM**) **antibody** titer by radioimmunoassay had been undetectable for more than 1 yr. Within 2 wk of the transplant, hematuria and **proteinuria** were noted; 5 mo. post-transplant renal biopsy showed linear IgG deposits in glomerular basement membrane and the **anti-GBM antibody** titer rose. Treatment with steroids, azathiopr[illegible] and [illegible]

Transplantation *11107
Movement 12100
Pathology, General and Miscellaneous - Inflammation and Inflammatory Disease 12508
Pathology, General and Miscellaneous - Therapy 12512
Metabolism - Carbohydrates 13004
Metabolism - Proteins, Peptides and Amino Acids *13012
Metabolism - Metabolic Disorders *13020
Cardiovascular System - Blood Vessel Pathology *14508
Blood, Blood-Forming Organs and Body Fluids - General; Methods *15001
Blood, Blood-Forming Organs and Body Fluids - Blood and Lymph Studies *15002
Urinary System and External Secretions - General; Methods 15501
Urinary System and External Secretions - Pathology *15506
Respiratory System - Pathology *16006
Endocrine System - Adrenals *17004
Pharmacology - Clinical Pharmacology 22005
Pharmacology - Endocrine System *22016
Pharmacology - Immunological Processes and Allergy *22018
Immunology and Immunochemistry - General; Methods 34502
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508

BC Hominidae 86215

IT Miscellaneous Descriptors
HUMAN STEROID AZATHIOPRINE IMMUNOLOGIC-DRUG GOODPASTURES SYNDROME IMMUNO **GLOBULIN** G HEMATURIA **PROTEINURIA** PLASMAPHERESIS

RN 446-86-6 (AZATHIOPRINE)

L17 ANSWER 14 OF 58 BIOSIS COPYRIGHT 2001 BIOSIS

AN 1979:226041 BIOSIS

DN BA68:28545

TI THE INTERACTION OF **ANTI** GLOMERULAR BASEMENT MEMBRANE **ANTIBODY** DEPOSITION WITH IMMUNE ELIMINATION OF BOVINE SERUM ALBUMIN IN THE RABBIT.

AU TREVILLIAN P; CAMERON J S

CS RENAL UNIT, DEP. MED., GUY'S HOSP. MED. SCH., LONDON SE1 9RT, ENGL., UK.

SO CLIN EXP IMMUNOL, (1979) 35 (3), 338-349.
CODEN: CEXIAL. ISSN: 0009-9104.

FS BA; OLD

LA English

AB The interaction of 2 different forms of immune glomerular damage occurring simultaneously were studied, i.e., **anti**-glomerular basement membrane (**GBM**) **antibody** fixation and immune elimination of bovine serum albumin (BSA). 125I-radiolabeled BSA **anti**-BSA immune complexes, formed in response to a single small i.v. dose (150 mg/kg) of 125I BSA, did not cause **proteinuria** in control animals within 15 days, despite evidence of immune elimination of the antigen. Similarly, a small dose of nephrotoxic **globulin** (NTG) (3.0 mg/kg) did not cause immediate **proteinuria** in controls. Test animals received the BSA injection followed by the NTG injection 5, 7 or 9 days later. In this way, **antibody** fixed to glomerular basement membrane antigens at various times after BSA **anti**-BSA complexes first appeared in the circulation. Animals were killed on day 15. Fifteen of the 18 test animals developed moderate to severe clinical **nephritis**. The onset of the **nephritis** coincided with BSA elimination irrespective of when the NTG was given. Greatly increased amounts of nonlinear immunofluorescent deposits were demonstrated in the glomeruli of test animals. There was a marked synergistic effect between 2 forms of immune glomerular damage (i.e., that mediated by **anti-GBM antibody** and immune complexes), which appeared to be due to the increased deposition of complex material in the presence of active fixation of **anti-GBM antibody**. The relevance of this finding to human glomerulonephritis was discussed.

CC Microscopy Techniques - Histology and Histochemistry 01056
Radiation - Radiation and Isotope Techniques 06504
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Biochemical Studies - Sterols and Steroids 10067
Biochemical Studies - Minerals 10069
Pathology, General and Miscellaneous - Inflammation and Inflammatory Disease 12508
Metabolism - Carbohydrates 13004

L17 ANSWER 15 OF 58 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1977:246829 BIOSIS
DN BA64:69193
TI STUDIES ON ACID ELUATES FROM KIDNEYS OF SHEEP WITH GLOMERULO **NEPHRITIS** MEDIATED BY **ANTIBODY** TO GLOMERULAR BASEMENT MEMBRANE.
AU BATSFORD S R; HARDWICKE J
SO INT ARCH ALLERGY APPL IMMUNOL, (1977) 54 (5), 475-478.
CODEN: IAAAAM. ISSN: 0020-5915.
FS BA; OLD
LA Unavailable
AB Kidneys from 6 sheep having glomerulonephritis mediated by **antibody** to glomerular basement membrane (**GBM**) were extracted at acid pH. Each preparation was characterized using immunological techniques and the eluates contained between 3.6 and 13% **anti-GBM antibody** of Ig[immunoglobulin]G class. This low **antibody** content is probably due to the presence of contaminants, mainly serum **proteins**.
CC Biochemical Methods - General 10050
Biochemical Studies - General 10060
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Biochemical Studies - Carbohydrates 10068
Biophysics - Membrane Phenomena 10508
Pathology, General and Miscellaneous - Inflammation and Inflammatory Disease 12508
Blood, Blood-Forming Organs and Body Fluids - Blood and Lymph Studies 15002
Urinary System and External Secretions - Pathology *15506
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
BC Bovidae 85715
IT Miscellaneous Descriptors
IMMUNO **GLOBULIN** G

L17 ANSWER 16 OF 58 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1977:198588 BIOSIS
DN BA64:20952
TI COMPLEMENT INDEPENDENT NEPHRO TOXIC **NEPHRITIS** IN THE GUINEA-PIG.
AU COUSER W G; STILMANT M M; JERMANOVICH N B
SO KIDNEY INT, (1977) 11 (3), 170-180.
CODEN: KDYIA5. ISSN: 0085-2538.
FS BA; OLD
LA Unavailable
AB Immunologic mechanisms of **proteinuria** were investigated in guinea pigs (GP) injected with sheep antiserum (NTS) to GP glomerular basement membrane (**GBM**). Linear deposition of sheep .gamma.1 and .gamma.2 Ig[immunoglobulinG led to a prompt but transient (36 h) increase in albumin excretion from control values of 0.026 .+-. 0.013 mg/h to maximal values of 26.3 .+-. 12.1 mg/h at 6 h without detectable histologic or EM changes except for decreased staining for glomerular polyanion and epithelial cell foot process fusion. **GBM** permeability to anionic ferritin was not increased during **proteinuria**. **Anti-GBM antibody** deposits did not fix GP C3 [the 3rd complement component] or C4 in vivo or in vitro. NTS-induced **proteinuria** was the same in guinea pigs that were normal, > 95% depleted of C3 through C9, genetically deficient in C4, and depleted of circulating polymorphonuclear leukocytes (PMN). Prior administration of antihistamines, steroids, azathioprine, colchicine, indomethacin, heparin, aprotinin (Trasylol), and niridazole also failed to reduce **proteinuria**. Initial **proteinuria** subsided by 36 h, did not recur despite linear deposition of GP .gamma.1 and .gamma.2 after day 7, and could not be produced by large or repeated doses of rabbit or GP **antibody** to **GBM**-bound sheep **globulin**. In the GP nephrotoxic **nephritis** model, **anti-GBM antibody** deposits apparently mediate increased permeability to albumin by a currently undefined mechanism which is independent of complement, PMN and other known mediators of inflammation.
CC Microscopy Techniques - Electron Microscopy 01058
Cytology and Cytochemistry - Animal 02506
Genetics and Cytogenetics - Animal 03506
Biochemical Studies - General 10060
Biochemical Studies - Nucleic Acids, Purines, [illegible]

Pharmacology - Connective Tissue, Bone and Collagen - Acting Drugs 22012
Pharmacology - Immunological Processes and Allergy 22018
Toxicology - General; Methods and Experimental 22501
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
Chemotherapy - Antiparasitic Agents 38510
Plant Physiology, Biochemistry and Biophysics - Chemical Constituents 51522
Pharmacognosy and Pharmaceutical Botany 54000
BC Liliaceae 25345
Bovidae 85715
Leporidae 86040
Caviidae 86300
IT Miscellaneous Descriptors
IMMUNO **GLOBULIN** G LINEAR DEPOSITION **PROTEINURIA**
GLOMERULAR BASEMENT MEMBRANE PERMEABILITY COLCHICINE METAB-DRUG
POLYMORPHONUCLEAR LEUKOCYTES **ANTI** GLOMERULAR BASEMENT
MEMBRANE SHEEP **ANTI** SERUM RABBIT **ANTIBODY**
RN 64-86-8 (COLCHICINE)

L17 ANSWER 17 OF 58 CAPLUS COPYRIGHT 2001 ACS
AN 2000:106177 CAPLUS
DN 132:260811
TI Endogenous glucocorticoids modulate experimental **anti**-glomerular basement membrane glomerulonephritis
AU Leech, M.; Huang, X. R.; Morand, E. F.; Holdsworth, S. R.
CS Centre for Inflammatory Diseases, Monash Medical Centre, Clayton, 3168, Australia
SO Clin. Exp. Immunol. (2000), 119(1), 161-168
CODEN: CEXIAL; ISSN: 0009-9104
PB Blackwell Science Ltd.
DT Journal
LA English
CC 2-4 (Mammalian Hormones)
Section cross-reference(s): 15
AB The influence of endogenous glucocorticoids (GC) on glomerular injury was studied in a rat model of heterologous **anti**-glomerular basement membrane (**GBM**) glomerulonephritis (GN). Sprague-Dawley rats underwent adrenalectomy (ADX) or sham-operation 3 days prior to i.v. administration of both **nephritogenic** (100 .mu.g/g) and subnephritogenic (50 .mu.g/g) doses of sheep **anti**-rat **GBM globulin**. Administration of a subnephritogenic dose of **anti-GBM globulin** resulted in GN in adrenalectomized animals only. Similarly, ADX performed prior to administration of **anti-GBM** in the **nephritogenic** dose range resulted in exacerbation of GN compared with sham-operated animals (24 h **protein** excretion: 190.8 vs. 42.5 mg/24 h). In ADX animals receiving subnephritogenic doses of **anti-GBM** injury was manifested by abnormal **proteinuria** (62.7 mg/24 h), accumulation of neutrophils which peaked at 6 h (7.2 neutrophils per glomerular cross-section (neut/gcs)) and macrophage accumulation in glomeruli at 24 h (6.8 macrophages/gcs). Sham-adrenalectomized animals given the same dose of **anti-GBM globulin** developed minimal or no glomerular injury: urinary **protein** excretion (8.7 mg/24 h); neutrophils (0.2 neutrophils/gcs); macrophages (1.2 macrophages/gcs). The increased cellular recruitment to glomeruli in adrenalectomized animals was assocd. with glomerular endothelial P-selectin expression. P-selectin expression was not detected in sham-operated rats after **anti-GBM** injection. Complement deposition in glomeruli was minimal in both groups. Physiol. GC replacement of ADX rats receiving subnephritogenic-dose **anti-GBM** reversed the obsd. susceptibility to GN development, with urinary **protein** excretion (7.8) and no detectable P-selectin expression or leukocyte accumulation in glomeruli. These results suggest that endogenous GC modulate heterologous **anti-GBM nephritis** in rats and that this may be attributable, in part, to regulation of P-selectin expression.
ST glucocorticoid **antibody** glomerulus basement membrane glomerulonephritis
IT Selectins
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(P-; endogenous glucocorticoids modulate experimental **anti**

-glomerular basement membrane glomerulonephritis and involved mechanisms)

IT **Proteins**, general, biological studies
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(**proteinuria**; endogenous glucocorticoids modulation of exptl. **anti**-glomerular basement membrane glomerulonephritis and involved mechanisms)

RE.CNT 41
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L17 ANSWER 18 OF 58 CAPLUS COPYRIGHT 2001 ACS
AN 1997:806417 CAPLUS
DN 128:74222
TI Influence of endotoxin contamination on **anti-GBM antibody** induced glomerular injury in rats

AU Karkar, Ayman M.; Rees, Andrew J.
CS Renal Unit, Dep. of Med., Royal Postgraduate Med. Sch., Hammersmith Hosp., London, UK

SO Kidney Int. (1997), 52(6), 1579-1583
CODEN: KDYIA5; ISSN: 0085-2538
PB Blackwell Science, Inc.
DT Journal
LA English

CC 15-8 (Immunochemistry)

AB It is accepted that the main determinant of glomerular injury in exptl. nephrotoxic **nephritis** is the administered dose of **anti**-glomerular basement membrane (**GBM**) **antibody**. However, there are other factors that can enhance the severity of such injury including small doses of bacterial lipopolysaccharide (LPS). In the present study, we have assessed whether prepns. of **anti-GBM antibody** contaminated with different concns. of

practical implications in studying models of **nephritis** as our results show that the glomerular injury, which is usually considered to be a sole function of the mass of **antibody** bound to **GBM**, is profoundly influenced by minor endotoxin contamination of the **anti-GBM antibody**.

ST lipopolysaccharide glomerular basement membrane **antibody** **nephritis**

IT Basement membrane
Glomerular injury
Rat
(influence of endotoxin contamination on **anti-GBM** **antibody** induced glomerular injury in rats)

IT **Antibodies**
Bacterial lipopolysaccharides
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(influence of endotoxin contamination on **anti-GBM** **antibody** induced glomerular injury in rats)

IT **Nephritis**
(nephrotoxic; influence of endotoxin contamination on **anti-GBM antibody** induced glomerular injury in rats)

L17 ANSWER 19 OF 58 CAPLUS COPYRIGHT 2001 ACS
AN 1996:74840 CAPLUS
DN 124:164675
TI Butein ameliorates experimental **anti**-glomerular basement membrane (**GBM**) **antibody**-associated glomerulonephritis in rats. (1)
AU Hayashi, Kazumi; Nagamatsu, Tadashi; Honda, Soichiro; Suzuki, Yoshio
CS Faculty Pharmacy, Meijo Univ., Nagoya, 468, Japan
SO Jpn. J. Pharmacol. (1996), 70(1), 55-64
CODEN: JJPAAZ; ISSN: 0021-5198
DT Journal
LA English
CC 1-7 (Pharmacology)
AB Effects of butein on crescentic-type **anti**-glomerular basement membrane (**GBM**) **nephritis** in rats were investigated. When rats were treated with butein from 1 day after i.v. injection of **anti-GBM** serum, it inhibited the elevation of **protein** excretion into urine. In the butein-treated rats, cholesterol content in plasma was lower than that of the **nephritic** control rats. Histol. observation demonstrated that this agent suppressed the incidence of crescent formation, adhesion of capillary wall to Bowman's capsule and fibrinoid necrosis in the glomeruli. Furthermore, butein suppressed the accumulation of leukocytes, including CD4-pos. cells and CD8-pos. cells in the glomeruli. However, butein failed to suppress the prodn. of the **antibody** against rabbit .gamma.-**globulin** and the deposition of rat-IgG on the **GBM**. These results suggest that butein may be a useful medicine against rapidly progressive glomerulonephritis, which is characterized by severe glomerular lesions with diffuse crescents.
ST butein crescentic glomerulonephritis
IT Kidney, disease
(crescentic glomerulonephritis, butein ameliorates **anti**-glomerular basement membrane **antibody**-assocd. glomerulonephritis)
IT 487-52-5, Butein
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(butein ameliorates **anti**-glomerular basement membrane **antibody**-assocd. glomerulonephritis)

L17 ANSWER 20 OF 58 CAPLUS COPYRIGHT 2001 ACS
AN 1995:626798 CAPLUS
DN 123:102323
TI Effects of acteoside (TJC-160) on alteration of adhesion molecules in glomeruli of crescentic-type **anti-GBM** **nephritic** rats
AU Hattori, Tomohisa; Fukuda, Yumiko; Takemoto, Norito; Shindo, Shoichiro; Kawamura, Hideki; Nishimura, Hiroaki; Maruno, Masao
CS Tsumura Central Laboratories, Tsumura & Co. Institute of New Drug Research, Ami, 300-11, Japan
SO Ensho (1995), 15(2), 147-54
CODEN: ENSHEE; ISSN: 0389-4290

antibody against rabbit .gamma. **globulin**. These results indicate that the antinephritic effect of TJC-160 may be at least partly due to the inhibition of glomerular infiltration of certain leukocyte subsets and the expression of adhesion mols.

ST acteoside TJC160 **nephritis** adhesion mol

IT Leukocyte
(glomerular infiltration; in effects of acteoside in glomeruli of crescentic-type **anti-GBM nephritic** rats)

IT Glycoproteins, specific or class
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(ICAM-1 (intercellular adhesion mol. 1), effects of acteoside on alteration of adhesion mols. in glomeruli of crescentic-type **anti-GBM nephritic** rats)

IT Kidney, disease
(**nephritis**, effects of acteoside on alteration of adhesion mols. in glomeruli of crescentic-type **anti-GBM nephritic** rats)

IT 61276-17-3, Acteoside
RL: BAC (Biological activity or effector, except adverse); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(effects of acteoside on alteration of adhesion mols. in glomeruli of crescentic-type **anti-GBM nephritic** rats)

L17 ANSWER 21 OF 58 CAPLUS COPYRIGHT 2001 ACS

AN 1994:621547 CAPLUS

DN 121:221547

TI Acteoside, a component of Stachys sieboldii MIQ, may be a promising antinephritic agent. (2): Effect of acteoside on leukocyte accumulation in the glomeruli of **nephritic** rats

AU Hayashi, Kazumi; Nagamatsu, Tadashi; Ito, Miko; Hattori, Tomohisa; Suzuki, Yoshio

CS Dep. Pharmacology, Meijo Univ., Nagoya, 468, Japan

SO Jpn. J. Pharmacol. (1994), 66(1), 47-52
CODEN: JJPAAZ; ISSN: 0021-5198

DT Journal

LA English

CC 1-8 (Pharmacology)

AB We investigated the effect of acteoside in comparison with that of cyclosporin A on leukocyte accumulation in the glomeruli of rats with crescentic-type **anti**-glomerular basement membrane (**GBM**) **nephritis**. Acteoside given p.o. at a dose of 30 mg/kg once a day for 15 consecutive days after treatment with **anti-GBM** serum markedly suppressed the urinary **protein** as well as glomerular histol. changes. Acteoside given p.o. for 5 or 15 consecutive days markedly suppressed the accumulation of total leukocytes, ED-1-pos. cells (monocytes/macrophages), CD4-pos. cells, CD8-pos. cells, interleukin-2-receptor-pos. cells (activated T cells) and Ia-pos. cells in the glomeruli. These effects of cyclosporin A (20 mg/kg/day, p.o.) were also as potent as those of acetoside (30 mg/kg/day, p.o.). Cyclosporin A also strongly suppressed the elevation of plasma **antibody** level against rabbit .gamma.-**globulin**. However, in this dose, acetoside did not significantly suppress the **antibody** formation. It can be concluded from these results that acetoside may exert its antinephritic action by suppressing the accumulation of leukocytes in the glomeruli.

ST acteoside leukocyte glomerulus **nephritis**

IT **Antibodies**
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(formation; acteoside vs. cyclosporin effect on leukocyte glomerular accumulation and **antibody** formation in relation to antinephritic activity)

IT Kidney, disease
(**nephritis**, acteoside vs. cyclosporin suppression of leukocyte glomerular accumulation in relation to antinephritic activity)

IT 61276-17-3, Acteoside
RL: BAC (Biological activity or effector, except adverse); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(acteoside vs. cyclosporin suppression of leukocyte glomerular accumulation in relation to antinephritic activity)

L17 ANSWER 22 OF 58 CAPLUS COPYRIGHT 2001 ACS

antibody prodn. against rabbit-.gamma.-**globulin** in the plasma were lower than those of the **nephritic** control rats. Histol. observation demonstrated that this agent suppressed hypercellularity and the incidence of crescent formation, adhesion of capillary wall to Bowman's capsule and fibrinoid necrosis in the glomeruli. Furthermore, rat-IgG and C3 deposits on the **GBM** were significantly less in the ACT-treated group than in the control **nephritic** group. When the treatment was started from the 20th day after i.v. injection of **anti-GBM** serum, by which the disease had been established, ACT had similar effect on the **nephritic** rats as stated above. These results suggest that ACT may be useful medicine against rapidly progressive glomerulonephritis, which is characterized by severe glomerular lesions with diffuse crescents.

ST acteoside crescentic glomerulonephritis

IT Kidney, disease
(crescentic glomerulonephritis, acteoside prevention of, **antibody** prodn. and complement activation suppression in relation to)

IT 61276-17-3P, Acteoside
RL: PREP (Preparation)
(crescentic glomerulonephritis prevention by, **antibody** prodn. and complement activation suppression in relation to)

L17 ANSWER 23 OF 58 CAPLUS COPYRIGHT 2001 ACS

AN 1993:52120 CAPLUS

DN 118:52120

TI Studies on the antinephritic effects of plant components. (6): Antinephritic effects and mechanisms of phellodendrine (OB-5) on crescentic-type **anti-GBM nephritis** in rats. (2)

AU Hattori, Tomohisa; Furuta, Kazuya; Hayashi, Kazumi; Nagamatsu, Tadashi; Ito, Mikio; Suzuki, Yoshio

CS Fac. Pharm., Meijo Univ., Nagoya, 468, Japan

SO Jpn. J. Pharmacol. (1992), 60(3), 187-95
CODEN: JJPAAZ; ISSN: 0021-5198

DT Journal

LA English

CC 1-8 (Pharmacology)

AB Effects of phellodendrine (OB-5) on crescentic-type **anti-GBM nephritis** in rats and the cell no. of the various leukocyte subpopulations in the glomeruli of the **nephritic** rats were investigated. OB-5 at 25, 50 and 100 mg/kg, p.o. prevented the urinary **protein** excretion by the 19th day after i.v.-injection of **anti-GBM** serum. In the OB-5 treated rats, plasma cholesterol and creatinine contents were lower than those of the control rats throughout the 40-day exptl. period. Histopathol. observations demonstrated that OB-5 inhibited the incidence of crescent formation, adhesion and fibrinoid necrosis in the glomeruli by the 41st day. OB-5 did not affect the plasma **antibody** titer against rabbit gamma **globulin**. The increases in total leukocytes, macrophages, cytotoxic/suppressor T cells, Ia pos. cells, and IL-2 receptor pos. cells in the glomeruli in OB-5, 100 mg/kg-treated rats as well as those of the animals treated with azathioprine or cyclosporin A were lower than those of the **anti-GBM nephritic** control. These results indicate that OB-5 was effective in crescentic-type **anti-GBM nephritis** and the antinephritic mechanisms of this agent may be due to its ability to inhibit the proliferation or the migration of macrophages and cytotoxic T lymphocytes in the glomeruli.

ST antinephritic phellodendrine macrophage T lymphocyte; interleukin 2 pos cell antinephretic phellodendrine

IT Leukocyte
Macrophage
(phellodendrine effect on proliferation and migration of, in glomeruli, antinephritic activity in relation to)

IT Lymphocyte
(T-cell, cytotoxic, phellodendrine effect on proliferation and migration of, in glomeruli, antinephritic activity in relation to)

IT Kidney, disease
(crescentic glomerulonephritis, **anti-GBM** , treatment of, by phellodendrine (OB-5), IL-2 pos. cell proliferation inhibition in)

anti-glomerular basement membrane) **nephritis** in rats
AU Nagao, Toshiyuki; Hattori, Tomohisa; Ito, Mikio; Suzuki, Yoshio
CS Fac. Pharm., Meijo Univ., Japan
SO Jpn. J. Nephrol. (1991), 33(3), 247-56
CODEN: NJGKAU; ISSN: 0385-2385
DT Journal
LA Japanese
CC 2-9 (Mammalian Hormones)
Section cross-reference(s): 63
AB The antinephritic effects of Lipo PGE1 on crescentic-type **anti**-glomerular basement membrane (**anti-GBM**) **nephritis** were examd. in rats. Lipo PGE1, given i.v. twice a day at 20.apprx.80 g/kg from the day after the **anti-GBM** serum injection (the 1st day), remarkably inhibited the urinary **protein** excretion as well as glomerular histopathol. changes such as crescent formation, adhesion of capillary walls to Bowman's capsule, the fibrinoid necrosis. Lipo PGE1, at antinephritic doses, significantly inhibited the elevation of platelet aggregation in renal vein and the decrease of renal blood flow. In addn., Lipo PGE1 significantly inhibited the elevation of plasma **antibody** titer against rabbit .gamma.-**globulin** that apparently reduced the deposition of rat IgG in glomeruli. The results suggest that i.v. Lipo PGE1 may be useful for the treatment of rapidly progressive glomerulonephritis and this agent may mainly exert the antinephritic action by reducing the deposition of immune complex in glomeruli via the suppression of host **antibody** formation. Furthermore, the inhibition of platelet aggregation and the increase in renal blood flow by Lipo PGE1 may also in part be related to the antinephritic action of this agent.
ST lipo PGE1 **nephritis** inhibitor
IT Blood platelet
(aggregation of, in kidney, lipo-PGE1 inhibition of)
IT Kidney
(circulation of and platelet aggregation in, lipo-PGE1 decrease of)
IT Circulation
(of kidney, lipo-PGE1 decrease of)
IT **Antibodies**
RL: BIOL (Biological study)
(to .gamma.-**globulins**, lipo-PGE1 decrease of, in kidney **nephritis**)
IT Kidney, disease or disorder
(glomerulonephritis, lipo PGE1 inhibition of)
IT **Proteins**, biological studies
RL: BIOL (Biological study)
(metabolic disorders, **proteinuria**, lipo-PGE1 inhibition of, in kidney **nephritis**)
IT **Globulins**, biological studies
RL: BIOL (Biological study)
(.gamma.-, **antibodies** to, lipo-PGE1 decrease of, in kidney **nephritis**)
IT 745-65-3, PGE1
RL: BIOL (Biological study)
(emulsified form of, antinephritic activity of)

L17 ANSWER 25 OF 58 CAPLUS COPYRIGHT 2001 ACS
AN 1990:70757 CAPLUS
DN 112:70757
TI Antinephritic effects of PGE1 and thiaprostaglandin E1, TEI 5178 and TEI 6122, on crescentic-type **anti-GBM nephritis** in rats
AU Nagamatsu, Tadashi; Kojima, Junko; Ito, Mikio; Kondo, Nobuyuki; Suzuki, Yoshio
CS Fac. Pharm., Meijo Univ., Nagoya, 468, Japan
SO Jpn. J. Pharmacol. (1989), 51(4), 521-30
CODEN: JJPAAZ; ISSN: 0021-5198
DT Journal
LA English
CC 2-9 (Mammalian Hormones)
GI

crescentic-type **anti**-glomerular basement membrane (**GBM**) **nephritis** in rats were investigated. The test compds. were s.c. administered every day for 39 days after the injection of **anti-GBM** serum. PGE1 (2.0 mg/kg/day), I (0.25 or 0.5 mg/kg/day), and II (0.25 or 0.5 mg/kg/day) reduced urinary **protein** by 30-50% of that of the control at the late stage of **nephritis**. These test compds. also suppressed the increase of blood urea N and the development of alterations in the glomeruli by the 40th day. Both I (0.5 mg/kg/day) and II (0.5 mg/kg/day) suppressed the prodn. of **antibody** to rabbit .gamma.-**globulin** in **nephritic** rats. This was not the case with PGE1, however. In addnl. expts. to clarify the antinephritic mechanisms of the test compds., it was found that 15 min after one s.c. injection of PGE1 (1.0 mg/kg), I (0.5 mg/kg), or II (0.5 mg/kg), systolic blood pressure in the **nephritic** rats was transiently reduced by 50-60%. On the other hand, these test compds. augmented renal blood flow (20-50%) from 45 min after the injection. The relations between the antinephritic effect and these subsequent findings are discussed.

ST kidney **nephritis** thiaprostaglandin; prostaglandin antinephritic activity; **nephritis** thiaprostaglandin E1

IT Blood pressure
(in **nephritis**, PGE1 and thiaprostaglandins effect on)

IT Circulation
(of kidney, in **nephritis**, PGE1 and thiaprostaglandins effect on)

IT **Proteins**, biological studies
RL: BIOL (Biological study)
(of urine, in **nephritis**, PGE1 and thiaprostaglandins effect on)

IT Urine
(**proteins** of, in **nephritis**, PGE1 and thiaprostaglandins effect on)

IT Kidney
(glomerulus, histol. of, in **nephritis**, PGE1 and thiaprostaglandins effect on)

IT Kidney, disease or disorder
(**nephritis**, kidney function in, PGE1 and thiaprostaglandins effect on)

IT 745-65-3, PGE1 83009-96-5, TEI 5178 83058-69-9, TEI 6122
RL: PRP (Properties)
(antinephritic effects of)

L17 ANSWER 26 OF 58 CAPLUS COPYRIGHT 2001 ACS
AN 1980:39672 CAPLUS
DN 92:39672
TI Interaction of concanavalin A and **GBM** glycoprotein in vivo
AU Nagasawa, Toshihiko
CS Sch. Med., Kyorin Univ., Tokyo, Japan
SO Jpn. Med. Res. Found. Publ. (1979), 7(Glomerulonephritis), 39-51
CODEN: JMRPDC
DT Journal
LA English
CC 15-13 (Immunochemistry)

AB Fluorescein isothiocyanate-conjugated concanavalin A (Con A) stained kidney glomerular basement membrane (**GBM**), tubular basement membrane (TBM), blood vessel walls, and the cytoplasm of the proximal tubular cells. I.v. injection of Con A into rabbits or rats resulted in hematuria, glycosuria, and lysozymuria by 20 min. These changes peaked at 60 min and disappeared after 3 days. **Proteinuria** appeared by 10 days. The Con A was found in the **GBM** and TBM soon after the injection. By 1 h, less Con A was found in the **GBM** and TBM, whereas it was present in the proximal tubule cytoplasm. By 3 days, Con A was present only in the proximal tubule cytoplasm. Con A was bound to a serum .alpha.2-**globulin** prior to its binding to kidney tissue. The binding distribution of Con A in the kidney was similar to that previously obsd. for **anti-nephritogenic** glycoprotein **antibody**.

ST concanavalin kidney basement membrane interaction; glycoprotein kidney concanavalin interaction

IT Basement membrane
(binding of concanavalin A by kidney, **nephritogenic** glycoprotein in relation to)

concanavalin A in relation to)
IT 11028-71-0
RL: PROC (Process)
(binding of, by kidney, **nephritogenic** glycoprotein in relation to)
IT 50-99-7, biological studies 9001-63-2
RL: BIOL (Biological study)
(of urine, concanavalin A binding to kidney tissue in relation to)

L17 ANSWER 27 OF 58 CAPLUS COPYRIGHT 2001 ACS
AN 1973:503290 CAPLUS
DN 79:103290
TI Experimental glomerulonephritis in the guinea pig. I. Glomerular lesions associated with antiglomerular basement membrane **antibody** deposits
AU Couser, W. G.; Stilmant, M.; Lewis, E. J.
CS Pritzker Sch. Med., Univ. Chicago, Chicago, Ill., USA
SO Lab. Invest. (1973), 29(2), 236-43
CODEN: LAINAW
DT Journal
LA English
CC 14-4 (Mammalian Pathological Biochemistry)
AB Studies of nephrotoxic **nephritis** have demonstrated that **antibody** to glomerular basement membrane (**GBM**) induces exptl. glomerulonephritis through complement- and polymorphonuclear leukocyte-mediated mechanisms. The immunopathogenesis of **anti-GBM nephritis** was studied in guinea pigs actively immunized with human **GBM** in Freund's complete adjuvant. Animals injected with Freund's complete adjuvant alone served as controls. Of the immunized animals 30% developed heavy **proteinuria**, but all animals studied (17 **proteinuric** and 33 nonproteinuric) had intense renal linear deposits of IgG **anti-GBM antibody**. Some animals in each group also had circulating **anti-GBM antibodies**. The **antibody** deposits were composed largely of .gamma.2 with variable amts. of .gamma.1 and IgM. Small amts. of complement were deposited in 2/3 of the animals studied and did not correlate with the presence of **proteinuria**. Five animals had heavy **proteinuria** without detectable .beta.1C-**globulin** deposition. Deposited, circulating, and eluted **anti-GBM antibody** from both **proteinuric** and nonproteinuric animals did not fix complement in vitro. Histol., **proteinuric** animals had mild, focal glomerular changes without an inflammatory exudate and a marked decrease in glomerular Alcian Blue staining compared to nonproteinuric and control animals. The absence of complement deposits in some **proteinuric** animals, lack of correlation between complement deposits and **proteinuria**, failure of **anti-GBM antibody** to fix complement in vitro, and the bland nature of the glomerular lesion suggest that **anti-GBM antibodies** mediate glomerular damage in this model through complement-independent mechanisms. The histochem. data suggest that these mechanisms may involve alterations in glomerular sialoprotein.
ST glomerulus lesion **antibody** deposit; antiglomerular basement membrane **antibody**
IT Basement membrane
(**antibodies** to glomerular, deposits of, in glomerulonephritis)
IT Kidney, disease or disorder
(glomerulonephritis, from nephrotoxic serum, basement membrane **antibody** deposits in)
IT **Antibodies**
RL: BIOL (Biological study)
(to glomerular basement membrane, deposits of, in glomerulonephritis)

L17 ANSWER 28 OF 58 CAPLUS COPYRIGHT 2001 ACS
AN 1971:403286 CAPLUS
DN 75:3286
TI Experimental glomerulonephritis in unresponsive rabbits after termination of immunological tolerance
AU Hammer, Dietrich K.
CS Max-Planck-Inst. Immunobiol., Freiburg-Zaehringen, Ger.
SO Curr. Probl. Immunol., Bayer-Symp., 1st (1969), Meeting Date 1968, 258-63.

IT **Antibodies**
RL: FORM (Formation, nonpreparative)
(formation of, glomerulonephritis in relation to tolerance in)
IT Basement membranes
(immune tolerance to kidney, glomerulonephritis in relation to)

L17 ANSWER 29 OF 58 CAPLUS COPYRIGHT 2001 ACS
AN 1970:98554 CAPLUS
DN 72:98554
TI Characterization of human **anti**-glomerular basement membrane **antibodies** eluted from glomerulonephritic kidneys
AU McPhaul, J. J., Jr.; Dixon, Frank J.
CS Scripps Clin. and Res. Found., La Jolla, Calif., USA
SO J. Clin. Invest. (1970), 49(2), 308-17
CODEN: JCINAO
DT Journal
LA English
CC 13 (Immunochemistry)
AB Eluates from glomerulonephritic kidneys of nine patients with **anti**-glomerular basement membrane (**anti-GBM**)-mediated **nephritis** were studied to define their antigenic specificity and content of kidney-fixing **antibodies**. Five of these patients had Goodpasture's syndrome with pulmonary and renal involvement clin., 4 patients did not. All had in vivo fixation of IgG in the characteristic linear pattern by direct immunofluorescence, and eluted IgG fixed to normal human kidney sections. Eluates from kidneys of patients with Goodpasture's syndrome fixed more frequently to homologous nonglomerular renal and extrarenal antigenic sites and to heterologous **GBM** than did nonGoodpasture eluates over a 100-fold range of **antibody** concns.; both could be blocked by prior absorption with sol. **GBM** antigens. By radial immunodiffusion and pptn. tests, the content of IgG in the eluates was 2-20% of the total **protein** eluted. By paired label isotopic fixation studies with some of the eluates, the percentage of IgG that was kidney-fixing ranged from 0.6 to 23.4%. Although the in vivo fixation studies with radiolabeled eluates failed to indicate significant fixation to monkey lung, the observations define quant. as well as qual. differences between **anti-GBM** **antibody** populations mediating the Goodpasture syndrome compared to those causing glomerulonephritis without lung involvement.
ST glomerulonephritis **antibodies**; **antibodies** glomerulonephritis; **nephritis antibodies**
IT **Globulins**, immune
RL: BIOL (Biological study)
(G, to basement membranes, in **nephritis**)
IT Basement membranes
(**antibodies** to, in **nephritis**)
IT Kidneys, diseases or disorders
(basement membrane **antibodies** in, Goodpasture's syndrome in relation to)
IT **Antibodies**
RL: BIOL (Biological study)
(to basement membranes, in **nephritis**)

L17 ANSWER 30 OF 58 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 2000043628 EMBASE
TI Endogenous glucocorticoids modulate experimental **anti**-glomerular basement membrane glomerulonephritis.
AU Leech M.; Huang X.R.; Morand E.F.; Holdsworth S.R.
CS M. Leech, Centre for Inflammatory Diseases, Monash Medical Centre, Locked Bag no. 29, Clayton, Vic. 3168, Australia. Michelle.Leech@med.monash.edu.au
SO Clinical and Experimental Immunology, (2000) 119/1 (161-168).
Refs: 41
ISSN: 0009-9104 CODEN: CEXIAL
CY United Kingdom
DT Journal; Article
FS 005 General Pathology and Pathological Anatomy
026 Immunology, Serology and Transplantation
028 Urology and Nephrology
LA English
SL English
AB The influence of endogenous glucocorticoids (GC) on glomerular injury was studied in a rat model of heterologous anti-glomerular basement

globulin developed minimal or no glomerular injury: urinary **protein** excretion (8.7 .+-. 1.5 mg/24 h, P<0.001); neutrophils (0.2 .+-. 0.04 neutrophils/gcs, P<0.001); macrophages (1.2 .+-. 0.5 macrophages/gcs, P<0.001). The increased cellular recruitment to glomeruli in adrenalectomized animals was associated with glomerular endothelial P-selectin expression. P-selectin expression was not detected in sham-operated rats after **anti-GBM** injection. Complement deposition in glomeruli was minimal in both groups. Physiologic GC replacement of ADX rats receiving subnephritogenic-dose **anti-GBM** reversed the observed susceptibility to GN development, with urinary **protein** excretion (7.8 .+-. 1.12, P<0:005) and no detectable P-selectin expression or leucocyte accumulation in glomeruli. These results suggest that endogenous GC modulate heterologous **anti-GBM nephritis** in rats and that this may be attributable, in part, to regulation of P-selectin expression.

CT Medical Descriptors:
*membranous glomerulonephritis: ET, etiology
glomerulonephritis: ET, etiology
autoimmune disease: ET, etiology
neutrophil
proteinuria
complement system
hormonal regulation
disease activity
nonhuman
male
rat
animal model
controlled study
article
priority journal
Drug Descriptors:
*glucocorticoid: EC, endogenous compound
***PADGEM protein**
***glomerulus basement membrane antibody**

L17 ANSWER 31 OF 58 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 1998021783 EMBASE
TI Influence of endotoxin contamination on **anti-GBM antibody** induced glomerular injury in rats.
AU Karkar A.M.; Rees A.J.
CS Dr. A.M. Karkar, Department of Medicine, Royal Postgraduate Medical School, Hammersmith Hospital, Du Cane Road, London W12 0NN, United Kingdom
SO Kidney International, (1997) 52/6 (1579-1583).
Refs: 13
ISSN: 0085-2538 CODEN: KDYIA5
CY United States
DT Journal; Article
FS 028 Urology and Nephrology
037 Drug Literature Index
LA English
SL English
AB It is accepted that the main determinant of glomerular injury in experimental nephrotoxic **nephritis** is the administered dose of **anti**- glomerular basement membrane (**GBM**) **antibody**. However, there are other factors that can enhance the severity of such injury including small doses of bacterial lipopolysaccharide (LPS). In the present study, we have assessed whether preparations of **anti-GBM antibody** contaminated with different concentrations of endotoxin could influence the severity of glomerular injury in the heterologous phase of nephrotoxic **nephritis**. We have also examined the efficacy of different laboratory methods to isolate an endotoxin-free **anti-GBM antibody**, and to purify **anti-GBM antibody** preparations from endotoxin. Preparations of **anti-GBM antibody** (nephrotoxic **globulin**) isolated from nephrotoxic serum by the sodium sulphate precipitation method contained variable concentrations of endotoxin Administration of these preparations in equal doses into clean rats, which had no established acute phase response, markedly aggravated the severity of glomerular injury. However, preparations contained less than 50 pg/ml of endotoxin appeared to have no significant effect on such injury.

antigen binding
reproducibility
immune response
affinity chromatography
nonhuman
rat
animal experiment
animal model
controlled study
article
priority journal
Drug Descriptors:
*endotoxin: CR, drug concentration
***glomerulus basement membrane antibody: CR, drug concentration**
alpha 1 microglobulin
bacterium lipopolysaccharide

L17 ANSWER 32 OF 58 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 97111089 EMBASE
DN 1997111089
TI **Antibody** independent crescentic glomerulonephritis in .mu. chain deficient mice.
AU Li S.; Holdsworth S.R.; Tipping P.G.
CS Dr. P. Tipping, Department of Medicine, Monash Medical Center, Clayton, Vic. 3168, Australia
SO Kidney International, (1997) 51/3 (672-678).
Refs: 29
ISSN: 0085-2538 CODEN: KDYIA5
CY United States
DT Journal; Article
FS 005 General Pathology and Pathological Anatomy
028 Urology and Nephrology
LA English
SL English
AB The hypothesis that crescent formation in glomerulonephritis (GN) is a delayed type hypersensitivity (DTH)-like lesion, not dependent on a humoral immune response, was addressed using mice with deletion of the .mu. immunoglobulin heavy chain gene (.mu. chain deficient mice). Homozygous .mu. chain deficient mice do not develop mature B cells or produce immunoglobulin, but have intact cell mediated immunity. GN was induced in sensitized mice by a subnephritogenic dose of sheep **anti**-mouse **GBM globulin**. Heterozygous mice (.mu. chain +/-) demonstrated normal **antibody** and DTH responses to sheep **globulin** and developed a proliferative GN with **proteinuria** (6.4 .+-. 1.4 mg/24 hr), renal impairment (serum creatinine 32.6 .+-. 3.3 .mu.mol/liter) and crescents in 33 .+-. 2.4% of glomeruli, when this antigen was planted in their glomeruli. This lesion was demonstrated to be T cell dependent by in vivo T cell depletion. Homozygous .mu. chain deficient mice (-/-) also developed proliferative GN, histologically indistinguishable from +/- mice. **Proteinuria** (3.8 .+-. 1.0 mg/24 hr), renal impairment (serum creatinine 24.5 .+-. 3.4 .mu.mol/liter) and crescent formation (29 .+-. 2% of glomeruli) were no different from =/- mice. Mouse immunoglobulin was absent in their serum and glomeruli, however, cutaneous DTH to sheep **globulin** was identical to heterozygous mice. These results demonstrate that glomerular crescent formation and injury can occur independent of a humoral immune response to planted glomerular antigen and without glomerular deposition of autologous **antibody**. This strongly supports the hypothesis that crescent formation is a manifestation of DTH.
CT Medical Descriptors:
***immune complex nephritis: ET, etiology**
*rapidly progressive glomerulonephritis: ET, etiology
animal experiment
animal model
animal tissue
article
mouse
nonhuman
priority journal
Drug Descriptors:
immunoglobulin heavy chain: EC, endogenous compound
immunoglobulin mu chain: EC, endogenous compound

SL English
AB Effects of butein on crescentic-type **anti**-glomerular basement membrane (**GBM**) **nephritis** in rats were investigated. When rats were treated with butein from 1 day after i.v. injection of **anti-GBM** serum, it inhibited the elevation of **protein** excretion into urine. In the butein-treated rats, cholesterol content in plasma was lower than that of the **nephritic** control rats. Histological observation demonstrated that this agent suppressed the incidence of crescent formation, adhesion of capillary wall to Bowman's capsule and fibrinoid necrosis in the glomeruli. Furthermore, butein suppressed the accumulation of leukocytes, including CD4-positive cells and CD8-positive cells in the glomeruli. However, butein failed to suppress the production of the **antibody** against rabbit .gamma.-**globulin** and the deposition of rat-IgG on the **GBM**. These results suggest that butein may be a useful medicine against rapidly progressive glomerulonephritis, which is characterized by severe glomerular lesions with diffuse crescents.
CT Medical Descriptors:
*autoimmunity
*glomerulonephritis: DT, drug therapy
*glomerulus basement membrane
adhesion
animal experiment
animal model
antibody production
article
capillary wall
cholesterol blood level
controlled study
cytotoxic t lymphocyte
drug structure
helper cell
histopathology
hypercholesterolemia: CO, complication
hypercholesterolemia: DT, drug therapy
immune complex deposition
immune complex nephritis: DT, drug therapy
immunoglobulin blood level
kidney capsule
leukocyte
male
necrosis: CO, complication
necrosis: DT, drug therapy
nonhuman
oral drug administration
protein urine level
rat
drug therapy
Drug Descriptors:
*butein: DT, drug therapy
*butein: PD, pharmacology
cholesterol: EC, endogenous compound
complement: EC, endogenous compound
cyclosporin a: CM, drug comparison
dipyridamole: CM, drug comparison
immunoglobulin g antibody
protein: EC, endogenous compound
RN (butein) 21849-70-7, 487-52-5; (cholesterol) 57-88-5; (complement) 9007-36-7; (cyclosporin a) 59865-13-3, 63798-73-2; (dipyridamole) 58-32-2; (**protein**) 67254-75-5
CO Dainippon (Japan); Sigma (United States); Sandoz (Japan)

L17 ANSWER 34 OF 58 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 94336381 EMBASE
DN 1994336381
TI Acteoside, a component of Stachys sieboldii MIQ, may be a promising antinephritic agent (2): Effect of acteoside on leukocyte accumulation in the glomeruli of **nephritic** rats.
AU Hayashi K.; Nagamatsu T.; Ito M.; Hattori T.; Suzuki Y.
CS Department of Pharmacology, Faculty of Pharmacy, Meijo University, 150 Yogotoyama,Tenpaku-ku, Nagoya 468, Japan
SO Japanese Journal of Pharmacology, (1994) 66/1 (47-52).
ISSN: 0021-5198 CODEN: JJPAAZ

ED-1-positive cells (monocytes/macrophages), CD4-positive cells, CD8-positive cells, interleukin-2-receptor-positive cells (activated T cells) and Ia-positive cells in the glomeruli. These effects of cyclosporin A (20 mg/kg/day, p.o.) were also as potent as those of acteoside (30 mg/kg/day, p.o.). Cyclosporin A also strongly suppressed the elevation of plasma **antibody** level against rabbit .gamma.-**globulin**. However, in this dose, acteoside did not significantly suppress the **antibody** formation. It can be concluded from these results that acteoside may exert its antinephritic action by suppressing the accumulation of leukocytes in the glomeruli.

CT Medical Descriptors:
*glomerulus
*leukocyte
***nephritis**
animal cell
animal experiment
animal model
animal tissue
antibody production
article
controlled study
drug effect
drug potency
glomerulus basement membrane
histology
macrophage
male
monocyte
nonhuman
oral drug administration
protein urine level
rat
t lymphocyte activation
Drug Descriptors:
*acteoside: CM, drug comparison
*acteoside: DV, drug development
*acteoside: PD, pharmacology
antibody: EC, endogenous compound
cd4 antigen: EC, endogenous compound
cd8 antigen: EC, endogenous compound
cyclosporin a: CM, drug comparison
cyclosporin a: PD, pharmacology
immunoglobulin
interleukin 2 receptor: EC, endogenous compound

RN (acteoside) 61276-17-3; (cyclosporin a) 59865-13-3, 63798-73-2; (immunoglobulin) 9007-83-4

CO Tsumura juntendo (Japan); Sandoz (Japan)

L17 ANSWER 35 OF 58 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.

AN 94220867 EMBASE

DN 1994220867

TI Acteoside, a component of Stachys sieboldii MIQ, may be a promising antinephritic agent: Effect of acteoside on crescentic-type **anti-GBM nephritis** in rats.

AU Hayashi K.; Nagamatsu T.; Ito M.; Hattori T.; Suzuki Y.

CS Department of Pharmacology, Faculty of Pharmacy, Meijo University, 150 Yagotoyama,Tenpaku-ku, Nagoya 468, Japan

SO Japanese Journal of Pharmacology, (1994) 65/2 (143-151).
ISSN: 0021-5198 CODEN: JJPAAZ

CY Japan

DT Journal; Article

FS 028 Urology and Nephrology
030 Pharmacology
037 Drug Literature Index

LA English

SL English

AB Effects of acteoside (ACT) on crescentic-type **anti-GBM nephritis** in rats were investigated. When rats were treated with ACT from the 1st day after i.v. injection of **anti-GBM** serum, ACT inhibited the elevation of **protein** excretion into urine. In the ACT-treated rats, cholesterol and creatinine contents and **antibody** production against rabbit .gamma.-**globulin** in [illegible] of the nephritic control rats

article
capillary wall
cell adhesion
controlled study
drug effect
glomerulus
histology
kidney capsule
kidney necrosis
male
nonhuman
oral drug administration
protein urine level
rapidly progressive glomerulonephritis
rat
Drug Descriptors:
*acteoside: CM, drug comparison
*acteoside: DV, drug development
*acteoside: PD, pharmacology
azathioprine: CM, drug comparison
azathioprine: PD, pharmacology
cholesterol: EC, endogenous compound
creatinine: EC, endogenous compound
dipyridamole: CM, drug comparison
dipyridamole: PD, pharmacology
glomerulus basement membrane antibody: EC, endogenous compound
immunoglobulin
plant extract: PD, pharmacology
plant extract: DV, drug development
plant extract: CM, drug comparison
RN (acteoside) 61276-17-3; (azathioprine) 446-86-6; (cholesterol) 57-88-5; (creatinine) 19230-81-0, 60-27-5; (dipyridamole) 58-32-2; (immunoglobulin) 9007-83-4
CO Tsumura juntendo (Japan); Boehringer ingelheim (Germany); Sigma (United States)

L17 ANSWER 36 OF 58 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 93009052 EMBASE
DN 1993009052
TI Studies on the antinephritic effects of plant components (6): Antinephritic effects and mechanisms of phellodendrine (OB-5) on crescentic-type **anti-GBM nephritis** in rats (2).
AU Hattori T.; Furuta K.; Hayashi K.; Nagamatsu T.; Ito M.; Suzuki Y.
CS Department of Pharmacology, Faculty of Pharmacy, Meijo University, 150 Yagotoyama,Tenpaku-ku, Nagoya 468, Japan
SO Japanese Journal of Pharmacology, (1992) 60/3 (187-195).
ISSN: 0021-5198 CODEN: JJPAAZ
CY Japan
DT Journal; Article
FS 026 Immunology, Serology and Transplantation
028 Urology and Nephrology
030 Pharmacology
037 Drug Literature Index
LA English
SL English
AB Effects of phellodendrine (OB-5) on crescentic-type **anti-GBM nephritis** in rats and the cell number of the various leukocyte subpopulations in the glomeruli of the **nephritic** rats were investigated. OB-5 at 25, 50 and 100 mg/kg/day, p.o. prevented the urinary **protein** excretion by the 19th day after i.v.-injection of **anti-GBM** serum. In the OB-5-treated rats, plasma cholesterol and creatinine contents were lower than those of the control rats throughout the 40-day experimental period. Histopathological observations demonstrated that OB-5 inhibited the incidence of crescent formation, adhesion and fibrinoid necrosis in the glomeruli by the 41st day. OB-5 did not affect the plasma **antibody** titer against rabbit gamma **globulin**. The increases in total leukocytes, macrophages, cytotoxic/suppressor T cells, Ia positive cells, and IL-2 receptor positive cells in the glomeruli in OB-5, 100 mg/kg-treated rats as well as those of the animals treated with azathioprine or cyclosporin A were lower than those of the **anti-GBM**
[illegible] These results indicate that OB-5 was effective

controlled study
creatinine blood level
cytotoxic t lymphocyte
drug effect
drug mechanism
glomerulus
growth inhibition
histopathology
kidney necrosis: DT, drug therapy
kidney necrosis: PC, prevention
leukocyte count
macrophage
male
nonhuman
oral drug administration
priority journal
protein urine level
rat
suppressor cell
t lymphocyte
Drug Descriptors:
interleukin 2 receptor
***glomerulus basement membrane antibody**
*phellodendron amurense extract: CM, drug comparison
*phellodendron amurense extract: DT, drug therapy
*phellodendron amurense extract: PD, pharmacology
Ia antigen: EC, endogenous compound
azathioprine: CM, drug comparison
creatinine: EC, endogenous compound
cyclosporin a: CM, drug comparison
phellodendrine: CM, drug comparison
phellodendrine: DT, drug therapy
phellodendrine: PD, pharmacology
rabbit antiserum
unclassified drug
RN (azathioprine) 446-86-6; (creatinine) 19230-81-0, 60-27-5; (cyclosporin a) 59865-13-3, 63798-73-2
CO Tsumura juntendo (Japan); Sandoz (Germany); Sigma (United States)

L17 ANSWER 37 OF 58 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 90295890 EMBASE
DN 1990295890
TI Glomerulonephritis in renal transplantation.
AU Vangelista A.; Frasca G.M.; Martella D.; Bonomini V.
CS Institute of Nephropathy, St Orsola University Hospital, Via Massarenti 9,40138 Bologna, Italy
SO Nephrology Dialysis Transplantation, (1990) 5/SUPPL. 1 (42-46).
ISSN: 0931-0509 CODEN: NDTREA
CY Germany
DT Journal; Conference Article
FS 028 Urology and Nephrology
037 Drug Literature Index
LA English
SL English
AB Recurrent glomerulonephritis and de novo glomerulonephritis may develop in the graft after renal transplantation. Among 59 patients with a pathological diagnosis of glomerulonephritis as original renal disease, 12 (20.3%) showed recurrence of the original lesions in the graft. Two patients with hereditary **nephritis** developed **anti-GBM** disease (one patients in two grafts). The disease rapidly progressed to graft loss. A de novo membranous nephropathy was diagnosed in four patients whose original renal disease was not a glomerulonephritis. One patient had been treated with antilymphocyte **globulin**, another with captopril.
CT Medical Descriptors:
*glomerulonephritis: DI, diagnosis
*kidney disease: DI, diagnosis
***proteinuria**
adolescent
adult
hematuria
major clinical study
human

Tenpaku-cho, Tenpaku-ku, Nagoya 468, Japan
SO Japanese Journal of Pharmacology, (1989) 51/4 (521-530).
ISSN: 0021-5198 CODEN: JJPAAZ
CY Japan
DT Journal; Article
FS 005 General Pathology and Pathological Anatomy
026 Immunology, Serology and Transplantation
028 Urology and Nephrology
030 Pharmacology
037 Drug Literature Index
LA English
SL English
AB The antinephritic effects of PGE1, TEI-5178 and TEI-6122 on crescentic-type **anti**-glomerular basement membrane (**GBM**) **nephritis** in rats were investigated. The test compounds were subcutaneously administered every day for 39 days after the injection of **anti-GBM** serum. PGE1 (2.0 mg/kg/day), TEI-5178 (0.25 or 0.5 mg/kg/day) and TEI-6122 (0.25 or 0.5 mg/kg/day) significantly reduced urinary **protein** by 30 to 50% of that of the control at the late stage of **nephritis**. These test compounds also suppressed the increase of blood urea nitrogen and the development of alteration in the glomeruli by the 40th day. Both TEI-5178 (0.5 mg/kg/day) and TEI-6122 (0.5 mg/kg/day) significantly suppressed the production of **antibody** to rabbit-.gamma. **globulin** in **nephritic** rats. This was not the case with PGE1 however. In additional experiments to clarify the antinephritic mechanisms of the test compounds, it was found that 15 min after one subcutaneous injection of PGE1 (1.0 mg/kg), TEI-5178 (0.5 mg/kg) or TEI-6122 (0.5 mg/kg), systolic blood pressure in the **nephritic** rats was transiently reduced by 50 to 60%. On the other hand, these test compounds augmented renal blood flow (20-50%) from 45 min after the injection. The relationship between the antinephritic effect and these subsequent findings will be discussed.
CT Medical Descriptors:
***immune complex nephritis: DT, drug therapy**
animal model
histology
kidney blood flow
proteinuria
rat
systolic blood pressure
urea nitrogen blood level
animal experiment
nonhuman
male
subcutaneous drug administration
article
priority journal
Drug Descriptors:
***glomerulus basement membrane antibody**
*prostaglandin e1: PD, pharmacology
*prostaglandin e1: DT, drug therapy
*prostaglandin e1: DO, drug dose
*15 cyclohexyl 16,17,18,19,20 pentanor 7 thiaprostaglandin e1 methyl ester: PD, pharmacology
*15 cyclohexyl 16,17,18,19,20 pentanor 7 thiaprostaglandin e1 methyl ester: DT, drug therapy
*15 cyclohexyl 16,17,18,19,20 pentanor 7 thiaprostaglandin e1 methyl ester: DO, drug dose
17,20 dimethyl 7 thiaprostaglandin e1 methyl ester: PD, pharmacology
17,20 dimethyl 7 thiaprostaglandin e1 methyl ester: DT, drug therapy
17,20 dimethyl 7 thiaprostaglandin e1 methyl ester: DO, drug dose
RN (prostaglandin e1) 745-65-3; (15 cyclohexyl 16,17,18,19,20 pentanor 7 thiaprostaglandin e1 methyl ester) 83009-96-5; (17,20 dimethyl 7 thiaprostaglandin e1 methyl ester) 83058-69-9
CN (1) Tei 5178; (2) Tei 6122
CO (2) Teijin (Japan); Funakoshi (Japan)

L17 ANSWER 39 OF 58 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 89098557 EMBASE
DN 1989098557
TI Exaggerated glomerular albuminuria after cobra venom factor in **anti**-glomerular basement membrane disease.

disease. A single injection of CVF 24 h before the administration of heterologous nephrotoxic **globulin** (NTG) to Sprague-Dawley rats resulted in greatly increased albuminuria in some animals on the second day of this model. This phenomenon was reproducible and depended on the presence of circulating PMN and complement. We have previously shown that the administration of CVF on days 9 and 11 of the HgCl2 model in inbred Brown Norway rats, resulted in increased albuminuria in all animals at day 17 ($p < 0.05$). The administration of small amounts of CVF with consequent complement activation in **antibody**-mediated disease represents a model for the increased injury seen after infection in human disease.

CT Medical Descriptors:
*allergic glomerulonephritis
*complement activation
*glomerulus basement membrane
***immune complex nephritis**
***proteinuria**
animal model
histology
rat
animal experiment
nonhuman
priority journal
Drug Descriptors:
*cobrotoxin

RN (cobrotoxin) 12584-83-7, 8001-03-4

L17 ANSWER 40 OF 58 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 84035535 EMBASE
DN 1984035535
TI Crescentic type **nephritis** induced by **anti**-glomerular basement membrane (**GBM**) serum in rats.
AU Ito M.; Yamada H.; Okamoto K.; Suzuki Y.
CS Department of Pharmacology, Faculty of Pharmacy, Meijo University, Tenpaku-ku, Nagoya 468, Japan
SO Japanese Journal of Pharmacology, (1983) 33/6 (1145-1154).
CODEN: JJPAAZ
CY Japan
DT Journal
FS 037 Drug Literature Index
030 Pharmacology
028 Urology and Nephrology
LA English
AB An experimental model of crescentic type **nephritis** was established by immunizing rats that had been given an i.v. **nephritogenic** dose (0.4 ml/animal) of rabbit **anti**-rat glomerular basement membrane (**GBM**) serum [**anti-GBM** serum] with 5 mg of rabbit .gamma.-**globulin** in Freund's complete adjuvant, and the process of **nephritis** was investigated by means of biochemical, histopathological and immunopathological analyses. Rats treated with **anti-GBM** serum and then with rabbit .gamma.-**globulin** (group II) showed significantly high levels or a tendency for high levels of urinary **protein** content. N-acetyl-.beta.-glucosaminidase activity and plasmin-like activity from the 20th to the 40th day observations after the induction of **nephritis**, when compared to rats given **anti-GBM** serum alone (group I). On the 40th day, plasma urea nitrogen, cholesterol and fibrinogen levels were significantly higher in group II than in group I. Glomerular histopathogical examination on the 40th day revealed that the incidence and the degree of severity of crescent formation, adhesion of capillary walls to Bowman's capsule and fibrinoid degeneration were remarkably greater in group II than in group I. However, no significant difference was seen between both groups on the thickening of capillary wall and mesangial proliferation. Linear deposits of rabbit IgG and rat IgG along the capillary walls as well as fibrinogen-reactive material deposits in Bowman's capsular spaces were observed by the immunofluorescence technique in both groups. The deposition of fibrinogen-reactive material was considerably greater in group II than in group I. Moreover, the depositon of rat IgG was slightly greater in group II. These results suggest that the **nephritis** of group II closely resembles rapidly progressive glomerulonephritis in humans and thus seems to be an adequate experimental model for screening beneficial drugs on this type of **nephritis**.

CT Medical Descriptors:

RN (immunoglobulin g) 97794-27-9

L17 ANSWER 41 OF 58 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 83249182 EMBASE
DN 1983249182
TI Factors affecting severity of injury during nephrotoxic **nephritis** in rabbits.
AU VanZyl Smit R.; Rees A.J.; Peters D.K.
CS Dep. Med., R. Postgrad. Med. Sch., Hammersmith Hosp., London W12 0H5, United Kingdom
SO Clinical and Experimental Immunology, (1983) 54/2 (366-372).
CODEN: CEXIAL
CY United Kingdom
DT Journal
FS 026 Immunology, Serology and Transplantation
028 Urology and Nephrology
LA English
AB All 22 rabbits injected with sheep **globulin** containing high titres of **antibodies** to rabbit glomerular basement membrane (GMMB) - nephrotoxic **globulin** (NTG) - developed **antibodies** to sheep IgG. Despite this only 15 rabbits developed obvious autologous phase injury. Eleven days after injection of NTG titres of autologous **antibody** to sheep IgG were similar in rabbits with and without definite autologous phase injury but were detected earlier and rose significantly more rapidly in those with autologous phase injury. In experiments on heterologous phase injury after injection of NTG, binding of defined amounts of nephrotic **antibodies** (NTAb) to the **GBM** after bolus injection caused significantly more injury, assessed by **proteinuria**, than binding of similar amounts of NTAb after infusion of NTG over 3 h ($P<0.02$ Student's paired t-test). In in vitro experiments, aliquots of homogenized rabbit kidney taken 2 days after injection of NTG bound appreciable amounts of rabbit **anti** -sheep Ig whereas homogenates of kidneys taken 20 days after NTG showed no such binding. These results show that the rate of deposition of NTAb in kidney influences the severity of injury in heterologous and autologous phases of NTN and that antigenic sites or heterologous and autologous phases of NTN and that antigenic sites or heterologous IgG fixed to the **GBM** became saturated during the autologous phase of injury.
CT Medical Descriptors:
*autoimmunity
*glomerulonephritis
***nephrotoxic serum nephritis**
proteinuria
rabbit
kidney
nonhuman

L17 ANSWER 42 OF 58 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 78064901 EMBASE
DN 1978064901
TI Complement independent nephrotoxic **nephritis** in the guinea pig.
AU Couser W.G.; Stilmant M.M.; Jermanovich N.B.
CS Dept. Med., Boston Univ. Med. Cent., Boston, Mass., United States
SO Kidney International, (1977) 11/3 (170-180).
CODEN: KDYIA5
DT Journal
FS 028 Urology and Nephrology
005 General Pathology and Pathological Anatomy
026 Immunology, Serology and Transplantation
025 Hematology
LA English
AB Immunologic mechanisms of **proteinuria** were investigated in guinea pigs (GP) injected with sheep antiserum (NTS) to GP glomerular basement membrane (**GBM**). Linear deposition of sheep .gamma.1 and .gamma.2 IgG led to a prompt but transient (36 hr) increase in albumin excretion from control values of 0.026.+-.0.013 mg/hr to maximal values of 26.3.+-.12.1 mg/hr at 6 hr without detectable histologic or electron microscopic changes except for decreased staining for glomerular polyanion and epithelial cell foot process fusion. **GBM** permeability to anionic ferritin was not increased during **proteinuria**. **Anti-GBM antibody** deposits did not fix GP C3 or C4 in vivo or in vitro. NTS-induced **proteinuria** was the same [illegible] of C3 through C9

cytology
electron microscopy
histology
diagnosis
etiology
Drug Descriptors:
*alloantibody
*complement
***glomerulus basement membrane antibody**
RN (complement) 9007-36-7

L17 ANSWER 43 OF 58 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 75144718 EMBASE
DN 1975144718
TI The significance of the glomeruli bound antirenal basement membrane active **antibody** as the pathogenetic factor of human chronic glomerulonephritis.
AU Masugi Y.; Sugisaki Y.; Ishizaki M.
CS Dept. Pathol., Nippon Med. Sch., Tokyo, Japan
SO Acta Pathologica Japonica, (1974) 24/5 (633-650).
CODEN: APJAAG
DT Journal
FS 005 General Pathology and Pathological Anatomy
028 Urology and Nephrology
026 Immunology, Serology and Transplantation
LA English
AB Acidic citric buffer eluates of the renal basement membranes (RBMs) purified from kidneys obtained at autopsies and corresponding sera of 7 cases of chronic glomerulonephritis (CGN), one case of Alport's syndrome and 22 other renal or non renal disease cases were examined immunopathologically. The renal eluates from all cases contained a certain amount of immunoglobulins especially IgG, the quantities of which were roughly parallel with the morphologic activities of glomerular changes. Most renal eluates from CGN cases showed not only in vitro **anti** RBM **antibody** activity (Boyden's method of passive hemagglutination) against the trypsin or collagenase digested and solubilized human RBM, but also in vivo glomerulonephritis producing capacity to rat kidneys with mobilization of complement fraction to the glomerular basement membrane (**GBM**) after i.v. administration. A considerable number of human CGN cases might be caused by **anti** RBM active autoantibody, which might have been produced in the bodies and fixed to the RBM (especially to the **GBM**) conducting initiation and progression of the course of the CGN cases. As to the antigenic determinant(s) of RBM against **anti** RBM **antibody**, it was suspected that **protein** or polypeptide moiety of RBM constituents plays a more important role than polysaccharide moiety of glycoprotein or glycopeptide.
CT Medical Descriptors:
***antigen antibody complex**
*chronic glomerulonephritis
*glomerulonephritis
*glomerulus basement membrane
*hemagglutination
***nephritis**
*kidney disease
major clinical study
autopsy
methodology
etiology
Drug Descriptors:
***antibody**
*autoantibody
***basement membrane antibody**
***beta globulin**
*complement
***glomerulus basement membrane antibody**
*glycoprotein
*immunoglobulin g
*immunoglobulin m
RN (beta **globulin**) 9007-02-7; (complement) 9007-36-7; (immunoglobulin g) 97794-27-9; (immunoglobulin m) 9007-85-6

L17 ANSWER 44 OF 58 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.

experimental glomerulonephritis through complement and polymorphonuclear leukocyte mediated mechanisms. Recent observations suggest that glomerular damage induced by **anti GBM antibody** may also be mediated through other mechanisms. The immunopathogenesis of **anti GBM nephritis** was studied in guinea pigs actively immunized with human **GBM** in complete Freund's adjuvant. Renal tissue, serum samples, and eluates were studied by routine histologic and immunofluorescent techniques. Animals injected with complete Freund's adjuvant alone served as controls. Thirty per cent (25/85) of immunized animals developed heavy **proteinuria**, but all animals studied (17 **proteinuric** and 33 nonproteinuric) had intense linear deposits of IgG **anti GBM antibody** documented by elution studies. Some animals in each group also had circulating **anti GBM antibodies**. The **antibody** deposits were composed largely of .gamma.2 with variable amounts of .gamma.1 and IgM. Small amounts of complement were deposited in two thirds of the animals studied and did not correlate with the presence of **proteinuria**. Five animals had heavy **proteinuria** without detectable .beta.1C **globulin** deposition. Furthermore, deposited, circulating, and eluted **anti GBM antibody** from both **proteinuric** and nonproteinuric animals did not fix complement in vitro. Histologically, **proteinuric** animals had mild, focal glomerular changes without an inflammatory exudate and a marked decrease in glomerular Alcian Blue staining compared to nonproteinuric and control animals. The absence of complement deposits in some **proteinuric** animals, lack of correlation between complement deposits and **proteinuria**, failure of **anti GBM antibody** to fix complement in vitro, and the bland nature of the glomerular lesion suggest that **anti GBM antibodies** mediate glomerular damage in this model through complement independent mechanisms. The histochemical data suggest that these mechanisms may involve alterations in glomerular sialoprotein.

CT Medical Descriptors:
*allergic glomerulonephritis
*autoimmune disease
*glomerulonephritis
*glomerulus
*glomerulus basement membrane
*immunofluorescence
*immunoglobulin g deposition
***proteinuria**
theoretical study
guinea pig
histology
cytology
methodology
Drug Descriptors:
***antibody**
*complement
***glomerulus basement membrane antibody**
*sialoprotein

RN (complement) 9007-36-7

L17 ANSWER 45 OF 58 CANCERLIT
AN 97148897 CANCERLIT
DN 97148897
TI Th1 responsiveness to **nephritogenic** antigens determines susceptibility to crescentic glomerulonephritis in mice.
AU Huang X R; Tipping P G; Shuo L; Holdsworth S R
CS Monash University, Department of Medicine, Monash Medical Centre, Clayton, Victoria, Australia.
SO KIDNEY INTERNATIONAL, (1997). Vol. 51, No. 1, pp. 94-103.
Journal code: KVB. ISSN: 0085-2538.
DT Journal; Article; (JOURNAL ARTICLE)
FS MEDL; L; Priority Journals
LA English
OS MEDLINE 97148897
EM 199705
AB The pattern of glomerulonephritis (GN) developing in response to a planted antigen (sheep **anti**-mouse **GBM globulin**) was compared in two strains of mice which demonstrated either a predominant Th1 (C57BL/6) or Th2 (BALB/c) response to this antigen. GN was induced

dependent. Treatment with monoclonal **anti**-mouse IFN gamma **antibody** significantly reduced glomerular injury and crescent formation and attenuated the cutaneous DTH response. GN induced by the same protocol in BALB/c mice exhibited pronounced glomerular IgG and complement deposition. Crescent formation, fibrin deposition, and glomerular T cell and macrophage infiltration were significantly less than observed in C57BL/6 mice, and injury was not T cell dependent in the effector phase. These data suggest that the pattern of glomerular injury induced by a planted antigen can be determined by the balance of T helper cell subset activation. A Th1 response induces a severe crescentic pattern of GN, which like cutaneous DTH, is T helper cell and IFN gamma dependent.

CT Check Tags: Animal; Male; Support, Non-U.S. Gov't
Antigens: IM, immunology
Antigens: PD, pharmacology
Antigens, CD4: IM, immunology
Autoantibodies: IM, immunology
Complement: AN, analysis
Creatinine: BL, blood
Creatinine: UR, urine
Fibrin: IM, immunology
Globulins: IM, immunology
*Glomerulonephritis: IM, immunology
Glomerulonephritis: PA, pathology
Hypersensitivity, Delayed: IM, immunology
IgG: IM, immunology
IgG: PD, pharmacology
Immunoglobulins, Intravenous
Interferon Type II: IM, immunology
Mice
Mice, Inbred BALB C
Mice, Inbred C57BL
Proteinuria
Sheep
*T-Lymphocytes, Helper-Inducer: IM, immunology

RN 60-27-5 (Creatinine); 82115-62-6 (Interferon Type II); 9001-31-4 (Fibrin); 9007-36-7 (Complement)

CN 0 (Antigens); 0 (Antigens, CD4); 0 (Autoantibodies); **0 (Globulins)**; 0 (IgG); 0 (Immunoglobulins, Intravenous)

L17 ANSWER 46 OF 58 CANCERLIT

AN 95056707 CANCERLIT

DN 95056707

TI Acetoside, a component of Stachys sieboldii MIQ, may be a promising antinephritic agent: effect of acteoside on crescentic-type **anti -GBM nephritis** in rats.

AU Hayashi K; Nagamatsu T; Ito M; Hattori T; Suzuki Y

CS Department of Pharmacology, Faculty of Pharmacy, Meijo University, Nagoya, Japan.

SO JAPANESE JOURNAL OF PHARMACOLOGY, (1994). Vol. 65, No. 2, pp. 143-51. Journal code: KO7. ISSN: 0021-5198.

DT Journal; Article; (JOURNAL ARTICLE)

FS MEDL; L; Priority Journals

LA English

OS MEDLINE 95056707

EM 199501

AB Effects of acetoside (ACT) on crescentic-type **anti-GBM nephritis** in rats were investigated. When rats were treated with ACT from the 1st day after i.v. injection of **anti-GBM** serum, ACT inhibited the elevation of **protein** excretion into urine. In the ACT-treated rats, cholesterol and creatinine contents and **antibody** production against rabbit gamma-**globulin** in the plasmas were lower than those of the **nephritic** control rats. Histological observation demonstrated that this agent suppressed hypercellularity and the incidence of crescent formation, adhesion of capillary wall to Bowman's capsule and fibrinoid necrosis in the glomeruli. Furthermore, rat-IgG and C3 deposits on the **GBM** were significantly less in the ACT-treated group than in the control **nephritic** group. When the treatment was started from the 20th day after i.v. injection of **anti-GBM** serum, by which the disease had been established, ACT resulted in a similar effect on the **nephritic** rats as stated above. These results suggest that ACT may be a useful medicine against rapidly progressive glomerulonephritis, which is characterized by severe glomerular lesions with diffuse crescents.

Immunosuppressive Agents: AD, administration & dosage
Immunosuppressive Agents: PD, pharmacology
*Immunosuppressive Agents: TU, therapeutic use
Kidney Glomerulus: DE, drug effects
Kidney Glomerulus: PA, pathology
Plant Extracts
Proliferating Cell Nuclear Antigen: ME, metabolism
Proteinuria: DT, drug therapy
Proteinuria: UR, urine
Rats
Rats, Sprague-Dawley
RN 57-88-5 (Cholesterol); 60-27-5 (Creatinine); 61276-17-3 (verbascoside)
CN 0 (Complement 3); **0 (Gamma-Globulins)**; 0 (Glucosides); 0 (Immunosuppressive Agents); 0 (Plant Extracts); 0 (Proliferating Cell Nuclear Antigen)

L17 ANSWER 47 OF 58 MEDLINE
AN 2000074847 MEDLINE
DN 20074847
TI Endogenous glucocorticoids modulate experimental **anti**-glomerular basement membrane glomerulonephritis.
AU Leech M; Huang X R; Morand E F; Holdsworth S R
CS Centre for Inflammatory Diseases, Monash Medical Centre, Clayton, Australia.. Michelle.Leech@med.monash.edu.au
SO CLINICAL AND EXPERIMENTAL IMMUNOLOGY, (2000 Jan) 119 (1) 161-8. Journal code: DD7. ISSN: 0009-9104.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 200004
EW 20000402
AB The influence of endogenous glucocorticoids (GC) on glomerular injury was studied in a rat model of heterologous **anti**-glomerular basement membrane (**GBM**) glomerulonephritis (GN). Sprague-Dawley rats underwent adrenalectomy (ADX) or sham-operation 3 days prior to i.v. administration of both **nephritogenic** (100 microgram/g) and subnephritogenic (50 microgram/g) doses of sheep **anti**-rat **GBM globulin**. Administration of a subnephritogenic dose of **anti-GBM globulin** resulted in GN in adrenalectomized animals only. Similarly, ADX performed prior to administration of **anti-GBM** in the **nephritogenic** dose range resulted in exacerbation of GN compared with sham-operated animals (24 h **protein** excretion: 190.8 +/- 32.8 versus 42.5 +/- 2.6 mg/24 h; P < 0.005). In ADX animals receiving subnephritogenic doses of **anti-GBM** injury was manifested by abnormal **proteinuria** (62.7 +/- 5.8 mg/24 h), accumulation of neutrophils which peaked at 6 h (7.2 +/- 1.37 neutrophils per glomerular cross-section (neut/gcs)) and macrophage accumulation in glomeruli at 24 h (6.8 +/- 1.2 macrophages/gcs). Sham-adrenalectomized animals given the same dose of **anti-GBM globulin** developed minimal or no glomerular injury: urinary **protein** excretion (8.7 +/- 1.5 mg/24 h, P < 0.001); neutrophils (0.2 +/- 0.04 neutrophils/gcs, P < 0.001); macrophages (1.2 +/- 0.5 macrophages/gcs, P < 0.001). The increased cellular recruitment to glomeruli in adrenalectomized animals was associated with glomerular endothelial P-selectin expression. P-selectin expression was not detected in sham-operated rats after **anti-GBM** injection. Complement deposition in glomeruli was minimal in both groups. Physiologic GC replacement of ADX rats receiving subnephritogenic-dose **anti-GBM** reversed the observed susceptibility to GN development, with urinary **protein** excretion (7.8 +/- 1.12, P < 0.005) and no detectable P-selectin expression or leucocyte accumulation in glomeruli. These results suggest that endogenous GC modulate heterologous **anti-GBM nephritis** in rats and that this may be attributable, in part, to regulation of P-selectin expression.
CT Check Tags: Animal; Male; Support, Non-U.S. Gov't
Adrenalectomy
Antibodies, Heterophile: AD, administration & dosage
Basement Membrane: IM, immunology
*Glomerulonephritis: ET, etiology
Glomerulonephritis: IM, immunology
Glomerulonephritis: PT, pathology

AU • Karkar A M; Rees A J
CS Department of Medicine, Royal Postgraduate Medical School, Hammersmith Hospital, London, England, United Kingdom.
SO KIDNEY INTERNATIONAL, (1997 Dec) 52 (6) 1579-83.
Journal code: KVB. ISSN: 0085-2538.
CY United States
DT Report; (TECHNICAL REPORT)
LA English
FS Priority Journals
EM 199803
EW 19980303
AB It is accepted that the main determinant of glomerular injury in experimental nephrotoxic **nephritis** is the administered dose of **anti**-glomerular basement membrane (**GBM**) **antibody**. However, there are other factors that can enhance the severity of such injury including small doses of bacterial lipopolysaccharide (LPS). In the present study, we have assessed whether preparations of **anti-GBM antibody** contaminated with different concentrations of endotoxin could influence the severity of glomerular injury in the heterologous phase of nephrotoxic **nephritis**. We have also examined the efficacy of different laboratory methods to isolate an endotoxin-free **anti-GBM antibody**, and to purify **anti-GBM antibody** preparations from endotoxin. Preparations of **anti-GBM antibody** (nephrotoxic **globulin**) isolated from nephrotoxic serum by the sodium sulphate precipitation method contained variable concentrations of endotoxin. Administration of these preparations in equal doses into clean rats, which had no established acute phase response, markedly aggravated the severity of glomerular injury. However, preparations contained less than 50 pg/ml of endotoxin appeared to have no significant effect on such injury. Furthermore, isolation of **anti-GBM antibody** from nephrotoxic serum by affinity chromatography, using Staphylococcus **protein**-A column, proved to be a reliable method not only for the isolation of an IgG (nephrotoxic **antibody**) free from other serum contaminants, but also for purification of endotoxin contaminated preparations of **anti-GBM antibody**. These observations have practical implications in studying models of **nephritis** as our results show that the glomerular injury, which is usually considered to be a sole function of the mass of **antibody** bound to **GBM**, is profoundly influenced by minor endotoxin contamination of the **anti-GBM antibody**.
CT Check Tags: Animal; Male; Support, Non-U.S. Gov't
***Antibodies: IP, isolation & purification**
Basement Membrane: IM, immunology
Charcoal
Chromatography, Affinity
*Endotoxins
*Kidney Glomerulus: IM, immunology
Nephritis: CI, chemically induced
***Nephritis: IM, immunology**
Polymyxin B
Rats
Rats, Sprague-Dawley
Staphylococcal Protein A
Sulfates
RN 1404-26-8 (Polymyxin B); 16291-96-6 (Charcoal); 7757-82-6 (sodium sulfate)
CN 0 (**Antibodies**); 0 (Endotoxins); 0 (Staphylococcal **Protein** A); 0 (Sulfates)

L17 ANSWER 49 OF 58 MEDLINE
AN 97148897 MEDLINE
DN 97148897
TI Th1 responsiveness to **nephritogenic** antigens determines susceptibility to crescentic glomerulonephritis in mice.
AU Huang X R; Tipping P G; Shuo L; Holdsworth S R
CS Monash University, Department of Medicine, Monash Medical Centre, Clayton, Victoria, Australia.
SO KIDNEY INTERNATIONAL, (1997 Jan) 51 (1) 94-103.
Journal code: KVB. ISSN: 0085-2538.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English

IFN gamma production by splenic T cells compared with C57BL/6 mice, consistent with a predominant Th2 response. In C57BL/6 mice, GN developing in response to sheep **globulin** exhibited a severe crescentic pattern with prominent glomerular T cell and macrophage influx and fibrin deposition. In vivo depletion with a monoclonal **anti**-CD4 **antibody** demonstrated that this injury was T helper cell dependent. Treatment with monoclonal **anti**-mouse IFN gamma **antibody** significantly reduced glomerular injury and crescent formation and attenuated the cutaneous DTH response. GN induced by the same protocol in BALB/c mice exhibited pronounced glomerular IgG and complement deposition. Crescent formation, fibrin deposition, and glomerular T cell and macrophage infiltration were significantly less than observed in C57BL/6 mice, and injury was not T cell dependent in the effector phase. These data suggest that the pattern of glomerular injury induced by a planted antigen can be determined by the balance of T helper cell subset activation. A Th1 response induces a severe crescentic pattern of GN, which like cutaneous DTH, is T helper cell and IFN gamma dependent.

CT Check Tags: Animal; Male; Support, Non-U.S. Gov't
Antigens: IM, immunology
Antigens: PD, pharmacology
Antigens, CD4: IM, immunology
Autoantibodies: IM, immunology
Complement: AN, analysis
Creatinine: BL, blood
Creatinine: UR, urine
Fibrin: IM, immunology
Globulins: IM, immunology
*Glomerulonephritis: IM, immunology
Glomerulonephritis: PA, pathology
Hypersensitivity, Delayed: IM, immunology
IgG: IM, immunology
IgG: PD, pharmacology
Immunoglobulins, Intravenous
Interferon Type II: IM, immunology
Mice
Mice, Inbred BALB C
Mice, Inbred C57BL
Proteinuria
Sheep
*T-Lymphocytes, Helper-Inducer: IM, immunology

RN 60-27-5 (Creatinine); 82115-62-6 (Interferon Type II); 9001-31-4 (Fibrin); 9007-36-7 (Complement)

CN 0 (Antigens); 0 (Antigens, CD4); 0 (Autoantibodies); 0 (**Globulins**); 0 (IgG); 0 (Immunoglobulins, Intravenous)

L17 ANSWER 50 OF 58 MEDLINE
AN 96419316 MEDLINE
DN 96419316
TI Butein ameliorates experimental **anti**-glomerular basement membrane (**GBM**) **antibody**-associated glomerulonephritis in rats (1).
AU Hayashi K; Nagamatsu T; Honda S; Suzuki Y
CS Department of Pharmacology, Meijo University, Nagoya, Japan.
SO JAPANESE JOURNAL OF PHARMACOLOGY, (1996 Jan) 70 (1) 55-64. Journal code: KO7. ISSN: 0021-5198.
CY Japan
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199701
EW 19970104
AB Effects of butein on crescentic-type **anti**-glomerular basement membrane (**GBM**) **nephritis** in rats were investigated. When rats were treated with butein from 1 day after i.v. injection of **anti-GBM** serum, it inhibited the elevation of **protein** excretion into urine. In the butein-treated rats, cholesterol content in plasma was lower than that of the **nephritic** control rats. Histological observation demonstrated that this agent suppressed the incidence of crescent formation, adhesion of capillary wall to Bowman's capsule and fibrinoid necrosis in the glomeruli. Furthermore, butein suppressed the accumulation of leukocytes, including CD4-positive cells and CD8-positive cells in the glomeruli. However, butein failed to

IgG: ME, metabolism
Kidney Glomerulus: DE, drug effects
*Kidney Glomerulus: IM, immunology
Kidney Glomerulus: PA, pathology
Leukocytes: DE, drug effects
Leukocytes: PA, pathology
Proteinuria: UR, urine
Rabbits
Rats
Rats, Sprague-Dawley
RN 487-52-5 (butein); 57-88-5 (Cholesterol); 94-41-7 (Chalcone)
CN 0 (**Antibodies**, Heterophile); 0 (IgG)

L17 ANSWER 51 OF 58 MEDLINE
AN 95165690 MEDLINE
DN 95165690
TI Acteoside, a component of Stachys sieboldii MIQ, may be a promising antinephritic agent (2): Effect of acteoside on leukocyte accumulation in the glomeruli of **nephritic** rats.
AU Hayashi K; Nagamatsu T; Ito M; Hattori T; Suzuki Y
CS Department of Pharmacology, Faculty of Pharmacy, Meijo University, Nagoya, Japan..
SO JAPANESE JOURNAL OF PHARMACOLOGY, (1994 Sep) 66 (1) 47-52.
Journal code: KO7. ISSN: 0021-5198.
CY Japan
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199505
AB We investigated the effect of acteoside in comparison with that of cyclosporin A on leukocyte accumulation in the glomeruli of rats with crescentic-type **anti**-glomerular basement membrane (**GBM**) **nephritis**. Acteoside given p.o. at a dose of 30 mg/kg once a day for 15 consecutive days after treatment with **anti-GBM** serum markedly suppressed the urinary **protein** as well as glomerular histological changes. Acteoside given p.o. for 5 or 15 consecutive days markedly suppressed the accumulation of total leukocytes, ED-1-positive cells (monocytes/macrophages), CD4-positive cells, CD8-positive cells, interleukin-2-receptor-positive cells (activated T cells) and Ia-positive cells in the glomeruli. These effects of cyclosporin A (20 mg/kg/day, p.o.) were also as potent as those of acteoside (30 mg/kg/day, p.o.). Cyclosporin A also strongly suppressed the elevation of plasma **antibody** level against rabbit gamma-**globulin**. However, in this dose, acteoside did not significantly suppress the **antibody** formation. It can be concluded from these results that acetoside may exert its antinephritic action by suppressing the accumulation of leukocytes in the glomeruli.
CT Check Tags: Animal; Comparative Study; Male
Cyclosporine: PD, pharmacology
Gamma-Globulins: IM, immunology
*Glomerulonephritis: DT, drug therapy
Glomerulonephritis: PA, pathology
*Glucosides: TU, therapeutic use
Immunohistochemistry
*Immunosuppressive Agents: TU, therapeutic use
*Kidney Glomerulus: PA, pathology
Leukocyte Count: DE, drug effects
*Leukocytes: DE, drug effects
*Plants, Medicinal: CH, chemistry
Proteinuria: DT, drug therapy
Rats
Rats, Sprague-Dawley
RN 59865-13-3 (Cyclosporine); 61276-17-3 (verbascoside)
CN 0 (Gamma-**Globulins**); 0 (Glucosides); 0 (Immunosuppressive Agents)

L17 ANSWER 52 OF 58 MEDLINE
AN 95056707 MEDLINE
DN 95056707
TI Acetoside, a component of Stachys sieboldii MIQ, may be a promising antinephritic agent: effect of acteoside on crescentic-type **anti-GBM nephritis** in rats.

hypercellularity and the incidence of crescent formation, adhesion of capillary wall to Bowman's capsule and fibrinoid necrosis in the glomeruli. Furthermore, rat-IgG and C3 deposits on the **GBM** were significantly less in the ACT-treated group than in the control **nephritic** group. When the treatment was started from the 20th day after i.v. injection of **anti-GBM** serum, by which the disease had been established, ACT resulted in a similar effect on the **nephritic** rats as stated above. These results suggest that ACT may be a useful medicine against rapidly progressive glomerulonephritis, which is characterized by severe glomerular lesions with diffuse crescents.

CT Check Tags: Animal; Male
Analysis of Variance
Antibody Formation
Cholesterol: BL, blood
Complement Hemolytic Activity Assay
Complement 3: ME, metabolism
Creatinine: BL, blood
Disease Models, Animal
Drug Screening
Gamma-Globulins: AD, administration & dosage
Gamma-Globulins: IM, immunology
*Glomerulonephritis: DT, drug therapy
Glomerulonephritis: IM, immunology
Glucosides: AD, administration & dosage
Glucosides: PD, pharmacology
*Glucosides: TU, therapeutic use
Immunohistochemistry
Immunosuppressive Agents: AD, administration & dosage
Immunosuppressive Agents: PD, pharmacology
*Immunosuppressive Agents: TU, therapeutic use
Kidney Glomerulus: DE, drug effects
Kidney Glomerulus: PA, pathology
Plant Extracts
Proliferating Cell Nuclear Antigen: ME, metabolism
Proteinuria: DT, drug therapy
Proteinuria: UR, urine
Rats
Rats, Sprague-Dawley

RN 57-88-5 (Cholesterol); 60-27-5 (Creatinine); 61276-17-3 (verbascoside)

CN 0 (Complement 3); 0 (Gamma-**Globulins**); 0 (Glucosides); 0 (Immunosuppressive Agents); 0 (Plant Extracts); 0 (Proliferating Cell Nuclear Antigen)

L17 ANSWER 53 OF 58 MEDLINE

AN 93148538 MEDLINE

DN 93148538

TI Studies on the antinephritic effects of plant components (6): antinephritic effects and mechanisms of phellodendrine (OB-5) on crescentic-type **anti-GBM nephritis** in rats (2).

AU Hattori T; Furuta K; Hayashi K; Nagamatsu T; Ito M; Suzuki Y

CS Department of Pharmacology, Faculty of Pharmacy, Meijo University, Nagoya, Japan..

SO JAPANESE JOURNAL OF PHARMACOLOGY, (1992 Nov) 60 (3) 187-95. Journal code: KO7. ISSN: 0021-5198.

CY Japan

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 199305

AB Effects of phellodendrine (OB-5) on crescentic-type **anti-GBM nephritis** in rats and the cell number of the various leukocyte subpopulations in the glomeruli of the **nephritic** rats were investigated. OB-5 at 25, 50 and 100 mg/kg/day, p.o. prevented the urinary **protein** excretion by the 19th day after i.v.-injection of **anti-GBM** serum. In the OB-5-treated rats, plasma cholesterol and creatinine contents were lower than those of the control rats throughout the 40-day experimental period. Histopathological observations demonstrated that OB-5 inhibited the incidence of crescent formation, adhesion and fibrinoid necrosis in the glomeruli by the 41st day. OB-5 did not affect the plasma **antibody** titer against rabbit gamma **globulin**. The increases in total leukocytes,

Glomerulonephritis: IM, immunology
Glomerulonephritis: PA, pathology
Immunohistochemistry
Kidney Glomerulus: IM, immunology
Proteinuria: UR, urine
*Quinolizines: TU, therapeutic use
Rats
Rats, Sprague-Dawley
RN 446-86-6 (Azathioprine); 57-88-5 (Cholesterol); 59865-13-3 (Cyclosporine); 60-27-5 (Creatinine); 6873-13-8 (phellodendrine)
CN 0 (**Antibodies**); 0 (**Antibodies**, Monoclonal); 0 (Quinolizines)

L17 ANSWER 54 OF 58 MEDLINE
AN 92349665 MEDLINE
DN 92349665
TI Suppression by cyclosporin A of **anti-GBM nephritis** in rats.
AU Nagamatsu T; Kojima N; Kondo N; Hattori T; Kojima R; Ito M; Suzuki Y
CS Department of Pharmacology, Faculty of Pharmacy, Meijo University, Nagoya, Japan..
SO JAPANESE JOURNAL OF PHARMACOLOGY, (1992 Jan) 58 (1) 27-36. Journal code: KO7. ISSN: 0021-5198.
CY Japan
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199211
AB The suppressive effect of cyclosporin A (CyA) on the development of glomerulonephritis was evaluated in rats with either original- or crescentic-type **anti**-glomerular basement membrane (**GBM**) **nephritis**. CyA (2.5, 10 or 20 mg/kg) was given p.o. daily to original-type **anti-GBM nephritic** rats for 10 days from the day after the injection of **anti-GBM** serum. The development of the **nephritis** was dose-dependently suppressed by CyA before the production of specific **antibody** against rabbit gamma-**globulin** (the heterologous phase). In addition, CyA suppressed glomerular infiltration of leukocyte subsets (leukocyte with common antigen, T cell, helper T cell, suppressor/cytotoxic T cell, macrophage/monocyte). CyA was given p.o. daily to crescentic-type **anti-GBM nephritic** rats for 10 days from the 10th day after the injection of **anti-GBM** serum. CyA-administration caused a distinct suppression of the deterioration of **nephritis** during the autologous phase. In addition, CyA markedly suppressed the **antibody** production. The above data indicate that CyA has a beneficial effect on **anti-GBM nephritis**, and the antinephritic action of this agent may be due to the inhibition of glomerular infiltration of leukocyte subsets as well as the suppression of the **antibody** production.
CT Check Tags: Animal; Male
Acetylglucosaminidase: UR, urine
Antibodies, Anti-Idiotypic: AN, analysis
Basement Membrane: IM, immunology
Cholesterol: BL, blood
Cyclosporine: AD, administration & dosage
*Cyclosporine: PD, pharmacology
Glomerulonephritis: IM, immunology
*Glomerulonephritis: PC, prevention & control
Immunosuppression
Kidney Glomerulus: IM, immunology
Leukocyte Count
Proteinuria: UR, urine
Rats
Rats, Inbred Strains
RN 57-88-5 (Cholesterol); 59865-13-3 (Cyclosporine)
CN EC 3.2.1.30 (Acetylglucosaminidase); 0 (**Antibodies, Anti-Idiotypic**)

L17 ANSWER 55 OF 58 MEDLINE
AN 91287218 MEDLINE
DN 91287218
TI Studies on antinephritic effect of lipo PGE1 (1). Effect of lipo PGE1 on crescentic type **anti-GBM nephritis** in rats.

Lipo PGE1 at doses, which the **anti-nephritic** action was recognized, significantly inhibited the elevation of platelet aggregation in renal vein and the decrease of renal blood flow. In addition, Lipo PGE1 significantly inhibited the elevation of plasma **antibody** titer against rabbit gamma-**globulin** the apparently reduced the deposition of rat IgG in glomeruli. These results suggest that intravenous Lipo PGE1 may be useful for the treatment of rapidly progressive glomerulonephritis and this agent may mainly exert the **anti-nephritic** action by reducing the deposition of immune complex in glomeruli via the suppression of host **antibody** formation. Furthermore, the inhibition of platelet aggregation and the increase in renal blood flow by Lipo PGE1 may be also in part related to the **anti-nephritic** action of this agent.

CT Check Tags: Animal; Male
Alprostadil: AD, administration & dosage
Alprostadil: PD, pharmacology
*Alprostadil: TU, therapeutic use
English Abstract
Glomerulonephritis: BL, blood
*Glomerulonephritis: DT, drug therapy
Glomerulonephritis: PA, pathology
Platelet Aggregation: DE, drug effects
Platelet Aggregation Inhibitors: PD, pharmacology
Rats
Rats, Inbred Strains
Renal Circulation

RN 745-65-3 (Alprostadil)

CN 0 (Platelet Aggregation Inhibitors)

L17 ANSWER 56 OF 58 MEDLINE

AN 90134497 MEDLINE

DN 90134497

TI Antinephritic effects of PGE1 and thiaprostaglandin E1, TEI-5178 and TEI-6122, on crescentic-type **anti-GBM** **nephritis** in rats.

AU Nagamatsu T; Kojima J; Ito M; Kondo N; Suzuki Y

CS Department of Pharmacology, Faculty of Pharmacy, Meijo University, Nagoya, Japan..

SO JAPANESE JOURNAL OF PHARMACOLOGY, (1989 Dec) 51 (4) 521-30.
Journal code: KO7. ISSN: 0021-5198.

CY Japan

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 199005

AB The antinephritic effects of PGE1, TEI-5178 and TEI-6122 on crescentic-type **anti**-glomerular basement membrane (**GBM**) **nephritis** in rats were investigated. The test compounds were subcutaneously administered every day for 39 days after the injection of **anti-GBM** serum. PGE1 (2.0 mg/kg/day), TEI-5178 (0.25 or 0.5 mg/kg/day) and TEI-6122 (0.25 or 0.5 mg/kg/day) significantly reduced urinary **protein** by 30 to 50% of that of the control at the late stage of **nephritis**. These test compounds also suppressed the increase of blood urea nitrogen and the development of alteration in the glomeruli by the 40th day. Both TEI-5178 (0.5 mg/kg/day) and TEI-6122 (0.5 mg/kg/day) significantly suppressed the production of **antibody** to rabbit gamma-**globulin** in **nephritic** rats. This was not the case with PGE1, however. In additional experiments to clarify the antinephritic mechanisms of the test compounds, it was found that 15 min after one subcutaneous injection of PGE1 (1.0 mg/kg), TEI-5178 (0.5 mg/kg) or TEI-6122 (0.5 mg/kg), systolic blood pressure in the **nephritic** rats was transiently reduced by 50 to 60%. On the other hand, these test compounds augmented renal blood flow (20-50%) from 45 min after the injection. The relationship between the antinephritic effect and these subsequent findings will be discussed.

CT Check Tags: Animal; Male
*Alprostadil: AA, analogs & derivatives
*Alprostadil: PD, pharmacology
***Antibodies**
Antibody Formation: DE, drug effects
Blood Pressure: DE, drug effects
Blood Urea Nitrogen

DN 84082780
TI Factors affecting severity of injury during nephrotoxic **nephritis** in rabbits.
AU Van Zyl Smit R; Rees A J; Peters D K
SO CLINICAL AND EXPERIMENTAL IMMUNOLOGY, (1983 Nov) 54 (2) 366-72. Journal code: DD7. ISSN: 0009-9104.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 198404
AB All 22 rabbits injected with sheep **globulin** containing high titres of **antibodies** to rabbit glomerular basement membrane (**GBM**)--nephrotoxic **globulin** (NTG)--developed **antibodies** to sheep IgG. Despite this only 15 rabbits developed obvious autologous phase injury. Eleven days after injection of NTG titres of autologous **antibody** to sheep IgG were similar in rabbits with and without definite autologous phase injury but were detected earlier and rose significantly more rapidly in those with autologous phase injury. In experiments on heterologous phase injury after intravenous injection of NTG, binding of defined amounts of nephrotoxic **antibodies** (NTAb) to the **GBM** after bolus injection caused significantly more injury, assessed by **proteinuria**, than binding of similar amounts of NTAb after infusion of NTG over 3 h (P less than 0.02 Student's paired t-test). In in vitro experiments, aliquots of homogenized rabbit kidney taken 2 days after injection of NTG bound appreciable amounts of rabbit **anti**-sheep Ig whereas homogenates of kidneys taken 20 days after NTG showed no such binding. These results show that the rate of deposition of NTAb in kidney influences the severity of injury in heterologous and autologous phases of NTN and that antigenic sites or heterologous IgG fixed to the **GBM** become saturated during the autologous phase of injury.
CT Check Tags: Animal; Support, Non-U.S. Gov't
***Antibodies, Anti-Idiotypic: BI, biosynthesis**
Complement 3: AN, analysis
Dose-Response Relationship, Immunologic
*IgG: IM, immunology
*Kidney Glomerulus: IM, immunology
Kidney Glomerulus: PA, pathology
***Nephritis: IM, immunology**
Nephritis: PA, pathology
Rabbits
Sheep: IM, immunology
Time Factors
CN 0 (**Antibodies, Anti**-Idiotypic); 0 (Complement 3)

L17 ANSWER 58 OF 58 MEDLINE
AN 79211909 MEDLINE
DN 79211909
TI The interaction of **anti**-glomerular basement membrane **antibody** deposition with immune elimination of bovine serum albumin in the rabbit.
AU Trevillian P; Cameron J S
SO CLINICAL AND EXPERIMENTAL IMMUNOLOGY, (1979 Mar) 35 (3) 338-49. Journal code: DD7. ISSN: 0009-9104.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 197911
AB We studied the interaction of two different forms of immune glomerular damage occurring simultaneously: **anti**-glomerular basement membrane (**GBM**) **antibody** fixation and immune elimination of bovine serum albumin (BSA). 125I-radiolabelled BSA **anti**-BSA immune complexes, formed in response to a single small intravenous dose (150 mg/kg) of 125I BSA, did not cause **proteinuria** in control animals within 15 days, despite evidence of immune elimination of the antigen. Similarly, a small dose of nephrotoxic **globulin** (NTG)(3.0 mg/kg) did not cause immediate **proteinuria** in controls. Test animals received the BSA injection followed by the NTG injection 5, 7 or 9 days later. In this way, **antibody** fixed to glomerular basement membrane antigens at various times after BSA **anti**-BSA complexes first appeared in the

Immunoenzyme Techniques
Kidney: PA, pathology
*Kidney Glomerulus: IM, immunology
Nephritis: IM, immunology
Nephritis: PA, pathology
Proteinuria: ET, etiology
Rabbits
*Serum Albumin, Bovine: IM, immunology

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(FILE 'HOME' ENTERED AT 10:32:01 ON 30 MAR 2001)

FILE 'BIOSIS, CAPLUS, EMBASE, CANCERLIT, MEDLINE' ENTERED AT 10:32:43 ON 30 MAR 2001

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L1            47 S ANTI-GMB
L2            23 S L1 AND NEPHRITIS
L3             0 S L2 AND (ALPHA2U GLOBULIN)
L4             0 S L2 AND (MAJOR URINARY PROTEIN)
L5             0 S L1 AND (MAJOR URINARY PROTEIN)
L6             0 S (MOUSE GLOMULAR BASAL MEMBRANE)
L7             2 S NAGAI/AU
L8          7090 S GBM
L9          1881 S L8 AND NEPHRIT?
L10         1404 S L9 AND ANTI
L11            0 S L10 AND (ALPHA GLOBULIN)
L12            0 S L10 AND (MAJOR URINARY PROTEIN)
L13          118 S L10 AND GLOBULIN?
L14           85 S L13 AND PROTEIN?
L15            0 S L14 AND ALPHA2
L16            0 S L14 AND FABP
L17           58 S L14 AND ANTIBOD?
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=> s l17 and kidney?

L18 36 L17 AND KIDNEY?

=> d l18 1-36 all